CT & MR IMAGING OF NEUROLOGICAL DISEASES IN PREGNANCY AND PUERPERIUM

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M.D. DEGREE

IN

RADIO DIAGNOSIS

DEPARTMENT OF RADIO DIAGNOSIS

COIMBATORE MEDICAL COLLEGE HOSPITAL

COIMBATORE

CERTIFICATE

This is to certify that the dissertation entitled **"CT & MR IMAGING OF NEUROLOGICAL DISEASES IN PREGNANCY AND PUERPERIUM"** is a record of bonafide work done by **DR.T.PRINCE JEBA ANAND**, Post graduate student in the Department of Radio Diagnosis, Coimbatore Medical College Hospital, Coimbatore.

Date :

Guide,
Professor, Department of Radio Diagnosis
Coimbatore Medical College & Hospital.

Date :

Professor & Head of the Department
Department of Radio Diagnosis
Coimbatore Medical College & Hospital

Date :

Dean,
Coimbatore Medical College & Hospital
Coimbatore.

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

(Affiliated to The Tamilnadu Dr. MGR Medical University, Chennai)

ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : PRINCE JEBA ANAND

Course : M.D RADIOLOGY.

Period of Study : 2013 - 2015

College : COIMBATORE MEDICAL COLLEGE.

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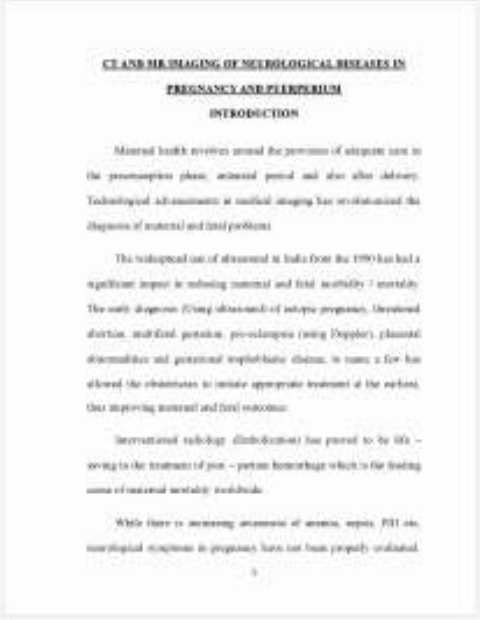


CT AND MR IMAGING OF NEUROLOGICAL DISEASES IN PREGNANCY AND PUERPERIUM

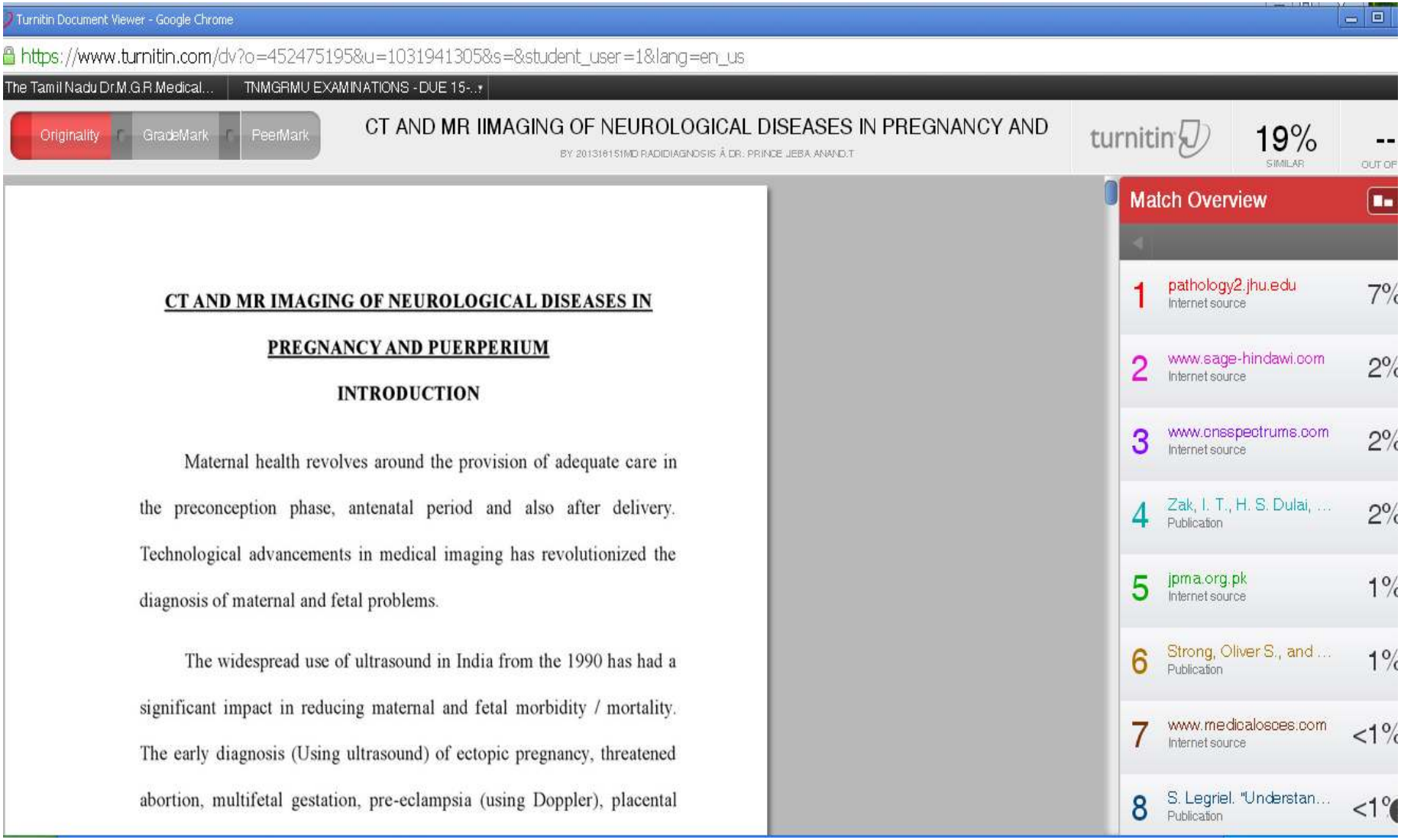
INTRODUCTION

Maternal health revolves around the provision of adequate care in the preconception phase, antenatal period and also after delivery. Technological advancements in medical imaging has revolutionized the diagnosis of maternal and fetal problems.

The widespread use of ultrasound in India from the 1990's has had a significant impact in reducing maternal and fetal morbidity / mortality. The early diagnosis (Using ultrasound) of ectopic pregnancy, threatened abortion, multifetal gestation, pre-eclampsia (using Doppler), placental abnormalities and gestational trophoblastic disease, in many a few has allowed the obstetricians to initiate appropriate treatment at the earliest, thus improving maternal and fetal outcomes.

Interventional radiology (Embolization) has proved to be life – saving in the treatment of post – partum hemorrhage which is the leading cause of maternal mortality worldwide.

While there is increasing awareness of anemia, sepsis, PIH etc, neurological symptoms in pregnancy have not been properly evaluated.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I solemnly declare that the dissertation titled "**CT AND MR IMAGING OF NEUROLOGICAL DISEASES IN PREGNANCY AND PUERPERIUM**" was done by me from 2013 onwards under the guidance and supervision of **Prof. Dr. N. Murali, M.D (RD).**

This dissertation is submitted to the Tamilnadu Dr.MGR Medical University towards the partial fulfillment of the requirement for the award of M.D Degree in Radio Diagnosis.

Place: **Dr. T. PRINCE JEBA ANAND**

Date:

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Date :

Place : Coimbatore **DR.T.PRINCE JEBA ANAND**

LIST OF ABBREVIATIONS USED

1.	CT	–	Computed Tomography.
2.	MRI	–	Magnetic Resonance Imaging.
3.	MRV	–	Magnetic Resonance Venography.
4.	DWI	–	Diffusion Weighted Images.
5.	PRES	–	Posterior Reversible Encephalopathy syndrome.
6.	PCA	–	Postpartum Cerebral Angiopathy.
7.	CVT	–	Cerebral Venous Thrombosis.
8.	SAH	–	Subarachnoid Hemorrhage.
9.	ICH	–	Intra Cerebral Hemorrhage.
10.	T1W	–	T1 weighted images
11.	T2W	–	T2 weighted images
12.	GRE	–	gradient echo sequence

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ABSTRACT

BACKGROUND

Pregnancy and puerperium is an important phase in medical sciences where unforseen circumstances can develop at any time. Some of the neurological signs and symptoms are indicative of impending risk while others are of no significance. This is where neuro imaging helps in identifying neurological disorders as well as in correlating them with the clinical presentations. Headache has been found to be clinically significant because it is usually associated with neurological disorders. So our objective is to establish the usefulness of imaging by CT and MRI in pregnancy and puerperium. This helps in advocating better treatment options and thereby have a good outcome.

OBJECTIVE

- To know the prevalence of disorders affecting the central nervous system during pregnancy and puerperium
- To understand the neuroimaging findings of the various neurological disorders in pregnancy and puerperium
- To evaluate the significance of CT &MR imaging in diagnosis neurological disorders in pregnancy and puerperium.
- To assess the usefulness of CT and MR imaging in determining the treatment modalities

MATERIALS

From August 2013 to august 2014, around 50 consecutive patients who presented with neurological signs and symptoms in Coimbatore Medical College Hospital, Coimbatore were selected for the study.

DESIGN: Observational study

SETTING: Department of Obstetrics and Gynaecology and Department of Radiodiagnosis.

STUDY METHOD: 50 consecutive patients who were selected included both pregnant women and those in the puerperium phase with neurological signs and symptoms.

History was elicited from these patients using the questionnaire that was framed for this purpose. Following this they were subjected to CT and MRI based on the indications. The prime motive is to make a relevant diagnosis based on imaging findings.

RESULTS

The study showed that cerebral venous thrombosis is the commonest neurological entity encountered in Coimbatore Medical College Hospital in pregnant women followed by Posterior reversible encephalopathy syndrome. This is quite contrary to the scenario in the

developed countries. It can be attributed to the environmental conditions, traditional practices as well as the socioeconomic status of the general population in developing countries like India.

Four cases of mortality with hemorrhagic infarcts due to venous thrombosis were recorded during the study period .Sudden onset of severe headache whether in antepartum women or young mothers in the immediate postpartum is the commonest symptom that correlated with positive imaging findings.

CONCLUSION Neurological symptoms are not uncommon during pregnancy and puerperium. While most symptoms turn out to be benign, in some patients they may indicate serious underlying problems. Use of prompt and appropriate imaging modality potentially helps to diagnose serious illness earlier and more accurately, thus helping the obstetrician to institute appropriate treatment strategies. This has a definite impact in reducing maternal morbidity and mortality

KEY WORD

Neurological disorders, pregnancy and puerperium, cerebral venous thrombosis.

INTRODUCTION

Maternal health revolves around the provision of adequate care in the preconception phase, antenatal period and also after delivery. Technological advancements in medical imaging have revolutionized the diagnosis of maternal and fetal problems.

The widespread use of ultrasound in India from the 1990's has had a significant impact in reducing maternal and fetal morbidity / mortality. The early diagnosis (using ultrasound) of ectopic pregnancy, threatened abortion, multifetal gestation, pre-eclampsia (using Doppler), placental abnormalities and gestational trophoblastic disease, to name a few has allowed the obstetrician to initiate appropriate treatment at the earliest, thus improving maternal and fetal outcomes.

Interventional radiology (Embolization) has proved to be life – saving in the treatment of postpartum hemorrhage which is the leading cause of maternal mortality worldwide.

While there is increasing awareness of anemia, sepsis, PIH etc, neurological symptoms in pregnancy have not been properly evaluated. Certain neurological complications that ensue during the

course of pregnancy can turn out to be disastrous, if not identified and treated early. The advent of CT and MRI has proved to be a boon in the early and accurate diagnosis of pregnancy related neurological complications. The study focuses on the non – invasive imaging evaluation of neurological symptoms occurring in pregnancy. By knowing the prevalence and spectrum of neurological complications in pregnancy, early appropriate treatment can be initiated, thus improving maternal outcomes.

OBJECTIVES

- To know the prevalence of disorders affecting the central nervous system during pregnancy and puerperium.
- To understand the neuroimaging findings of the various neurological disorders in pregnancy and puerperium.
- To evaluate the significance of CT &MR imaging in diagnosis of neurological disorders in pregnancy and puerperium.
- To assess the usefulness of CT and MR imaging in determining the treatment modalities.

REVIEW OF LITERATURE

A spectrum of pathologic disorders involve the central nervous system and pituitary gland in both pregnancy and puerperium. Few neurologic conditions that are related to the physiologic modifications of the reproductive system (for instance eclampsia, reversible cerebral vasoconstriction syndrome, sheehan syndrome).

Retrograde analysis based on clinical study helps to understand the pathologic variants and differentiate the neurological entities inspite of the inexplicable clinical signs and symptoms that are liable to occur in pregnancy and postpartum period.

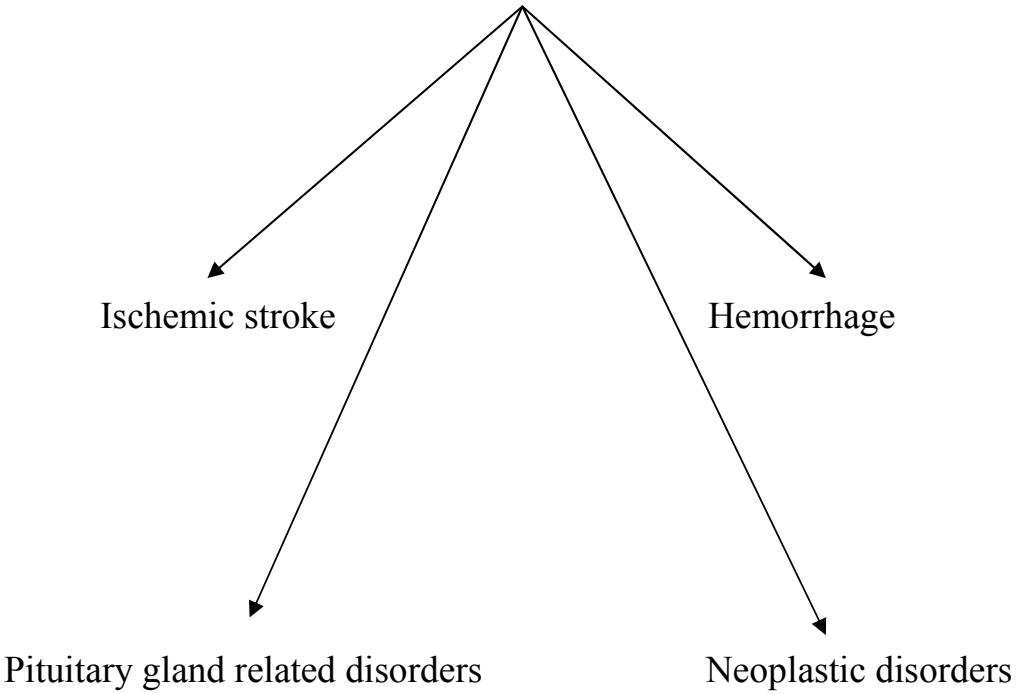
Some of the cerebrovascular diseases are nonspecific to pregnancy but occur more frequently in pregnancy and post partum women particularly, cerebral infarction, dural venous thrombosis and pituitary apoplexy. Most often, these conditions may have subtle presentations and go unnoticed.

But in terms of maternal mortality and morbidity, neuro-radiological imaging has rendered a major assistance in perspective of determining the treatment options as well as monitoring the prognosis.

Nevertheless clinicians are yet reluctant to let pregnant women undergo imaging –related investigations due to radiation hazards.

Cerebrovascular complications are distilled into four major categories of ischemic stroke, hemorrhage (subarachnoid hemorrhage, eclamptic encephalopathy, cerebral venous thrombosis), pituitary gland related disorders (pituitary apoplexy, pituitary adenoma, sheehans syndrome, lymphocystic adenohypophysitis) and other neoplastic disorders (Primary intracranial tumors and intracranial metastasis).

CEREBRO VASCULAR NEUROLOGICAL COMPLICATIONS IN PREGNANCY AND PUERPERIUM



BRAIN IMAGING DURING PREGNANCY

Imaging studies in pregnant women should be based on neurological indications but clinicians are reluctant due to concerns about fetal exposure to radiation. The harmful effects of radiation is determined by the stage of gestation at which the fetus is exposed, the total dose of radiation absorbed, and the rate at which the dose is absorbed.

Exposure of fetus to ionizing radiation from CT of the maternal head is extremely low. Fetal anomalies due to radiation are presumed to occur during the first few weeks, the embryogenesis period, when the patient may not be aware of the pregnancy.

Protective shielding for the developing fetus should be used whenever pregnancy is suspected. CT perfusion studies should be avoided due to a significant increase of the X-ray exposure. Also the use of iodinated intravenous contrast material is perilous to the fetus.

There is no documentation of hazardous fetal effects in humans to the magnetic field exposure for magnetic resonance imaging (MRI).

It has been postulated that there is a minimal risk for the fetus even with exposure to very powerful magnetic fields, minimal increases in body temperature, and loud tapping noises of the coils. If clinically feasible, MRI is the preferred imaging option in pregnancy

ISCHEMIC STROKE

Ischemic infarction of pregnancy and puerperium accounts for 60% of all strokes. Pregnancy and the puerperium bring about an alteration in the levels of inhibitors of coagulant proteins(1,2).

Inhibitors of protein S is lowered but inhibitors of protein C are elevated. There is increase in the levels of clotting factors fibrinogen, factor VII, factor VIII, and factor X.

In addition heparin neutralization capacity is increased. Protein S (total and free) tends to lower by 10 weeks of gestation and progressively decreases during the entire pregnancy(1).

Enhanced resistance to activated protein C that has been ascribed. It is seen in majority of the normal pregnant women than in normal non pregnant controls. However, absolute levels of protein C have no notable changes.

It may be elevated in the immediate postpartum period. Likewise antithrombin (AT) levels are unaltered. The Factor V after 16 weeks of gestation and the activity increases by 29% at term Fibrinogen levels start to rise (1)as early as 6-7 weeks and continue to rise thereafter.

From 20th week onwards prothrombin time shortens, probably as a result of increased levels of factor VII. Changes in protein S, resistance to APC, factors V and VIII, and fibrinogen persist during puerperium, at least a few days.

On the whole, the fibrinolytic system is impeded by increased PA1 activity during pregnancy (1,2). Platelets have a vital role in the coagulation. Though the platelet count is adequate there is more affinity for aggregation. Moreover there is less stimuli for prostacyclin and CAMP production.

These entire characteristic changes as well other molecular mediators of hemostasis shift the balance towards a hypercoagulant effect. Probabilities of obstetric stroke to be of venous origin is higher in comparison to stroke due to other factors.

Procoagulant states are more marked around term and worsens in the immediate postpartum period (4,12), presumably related to the expulsion of the placenta and release of thromboplastic substances following placental separation.

Blood coagulation and fibrinolysis switchover to those of the non pregnant state by around 3 weeks after delivery (3,4).

Changes in blood coagulation factors			
Parameters	***Non pregnant***	***Pregnancy near term***	***change***
Platelets (cu mm)	1,60,000 – 2,00,000	Conflicting observation	Static or 15% reduction of the count
Fibrinogen (mg%)	200 – 400	300 – 600	+50%
Fibrinolytic Activity	-	Depressed	-
Clotting Time	-	Unaffected	-
ESR	10 mm/h	40 mm/h`	Marked increase (4 times)

Although pregnancy-related stroke is known to have higher incidence among black women particularly those aged 35 years and older, ischemic subtype has been associated with a younger maternal age. Migraine headache have been associated with a 17-fold increased risk of stroke in pregnancy. Caesarean delivery has been shown to be associated with a 3–12 times heightened risk in peripartum and postpartum period(7,8).

Other risk factors include lupus and blood transfusion.

Atherosclerotic plaques in the setting of a hypercoagulable state eventually results in thrombotic infarct. Embolic infarcts classically occur in the major arterial branches. Obstructed labor can precipitate embolic episodes.

Dilated peripartum cardiomyopathy and valvular lesions in the heart are liable to cause infarction. Watershed infarcts occur due to excessive post partum bleeding.

Early diagnosis and intervention by administration of thrombolytic agents such as human tissue plasminogen activator (Rt-PA) is a life – saving procedure and has favorable maternal outcome(9). Due to its large molecular size, Rt-PA does not cross over to the fetus and pharmacologic studies do not demonstrate teratogenicity.

In pregnant women who are at potential risk for ischemic stroke, anticoagulant therapy is advocated with warfarin, unfractionated heparin and low molecular weight heparin. Low dose aspirin and clopidogrel are used as a prophylactic measures.

VENOUS DRAINAGE OF THE BRAIN

The veins draining the brain open into the dural venous sinuses. These are the superior sagittal, inferior sagittal, straight, transverse, sigmoid, cavernous, sphenoparietal, petrosal and occipital sinuses.

Ultimately, the blood from all these sinuses reaches the sigmoid sinus which becomes continuous with the internal jugular vein. The intracranial venous sinuses communicate with veins outside the skull through emissary veins.

The venous drainage of individual parts of the brain is described below.

Veins Of The Cerebral Hemisphere

The veins of the cerebral hemisphere consist of two sets,

1. Superficial.
2. Deep.

The superficial veins drain into neighbouring venous sinuses. The superior cerebral veins drain the upper parts of the superolateral and medial surfaces, and end in the superior sagittal sinus.

Some veins from the medial surface join the inferior sagittal sinus. Inferior cerebral veins drain the lower part of the hemisphere.

On the superolateral surface, they drain into the superficial middle cerebral vein which lies superficially along the lateral sulcus and its posterior ramus. The posterior end of this vein is connected to the superior sagittal sinus by the superior anastomotic vein; and to the transverse sinus by the inferior anastomotic vein.

The superficial middle cerebral vein terminates in the cavernous sinus. Veins from the inferior surface of the cerebral hemisphere drain into the transverse, superior petrosal, cavernous and sphenoparietal sinuses. Some may ascend to join the inferior sagittal sinus.

The deep veins of the cerebral hemisphere are the two internal cerebral veins, that joint to form the great cerebral vein and the two basal veins, that wind round the midbrain to end in the great cerebral vein.

Each internal cerebral vein begins at the interventricular foramen, and runs backwards in the tela choroidea, in the roof of the third ventricle. It has numerous tributaries. One of these is the thalamostriate vein which lies in the floor of the lateral ventricle (between the thalamus, medially; and the caudate nucleus, laterally).

Each basal vein begins near the anterior perforated substance. It is formed by union of the following.

(a). The anterior cerebral vein, which accompanies the anterior cerebral artery.

(b). The deep middle cerebral vein, which lies deep in the stem and posterior ramus of the lateral sulcus.

(c). Some inferior striate veins that emerge from the anterior perforated substance.

The great cerebral vein, formed by union of the two internal cerebral veins, passes posteriorly beneath the splenium of the corpus collosum, to end in the straight sinus. It receives the basal veins, some veins from the occipital lobes, and some from the corpus callosum.

The deep cerebral veins described above are responsible for draining the thalamus, the hypothalamus, the corpus striatum, the septum pellucidum, and the choroid plexuses.

Many of the cerebral vein tributaries extend beyond the corpus striatum into the myelinated segment of the cerebral hemispheres. Here they establish communications with superficial veins. They can thus serve as alternative channels for draining parts of the cerebral cortex.

The upper part of the thalamus is drained by the tributaries of the internal cerebral vein (Including the thalamostriate vein). The lower part of the thalamus, and the hypothalamus, are drained by veins that run downwards to end in a plexus of veins present in the interpeduncular fossa. This plexus drains into the cavernous and sphenoparietal sinuses, and into the basal veins.

The corpus striatum and internal capsule are drained by two sets of striate veins. The superior striate veins run dorsally and drain into tributaries of the internal cerebral vein. The inferior striate veins run vertically downwards and emerge on the base of the brain through the anterior perforated substance. Here they end in the basal vein.

VEINS OF THE CEREBELLUM AND BRAINSTEM

The veins from the upper surface of the cerebellum drain into the straight, transverse, and superior petrosal venous sinuses. Veins from the inferior surface drain into the right and left sigmoid and inferior petrosal, sinuses the occipital sinuses and the straight sinus.

The veins of the midbrain drain into the great cerebral veins or into the basal vein. The pons and medulla drain into the superior and inferior petrosal sinuses, the transverse sinus and the occipital sinus. Inferiorly, the veins of the medulla are continuous with the veins of the spinal cord.

THE INTRACRANIAL VENOUS SYSTEM

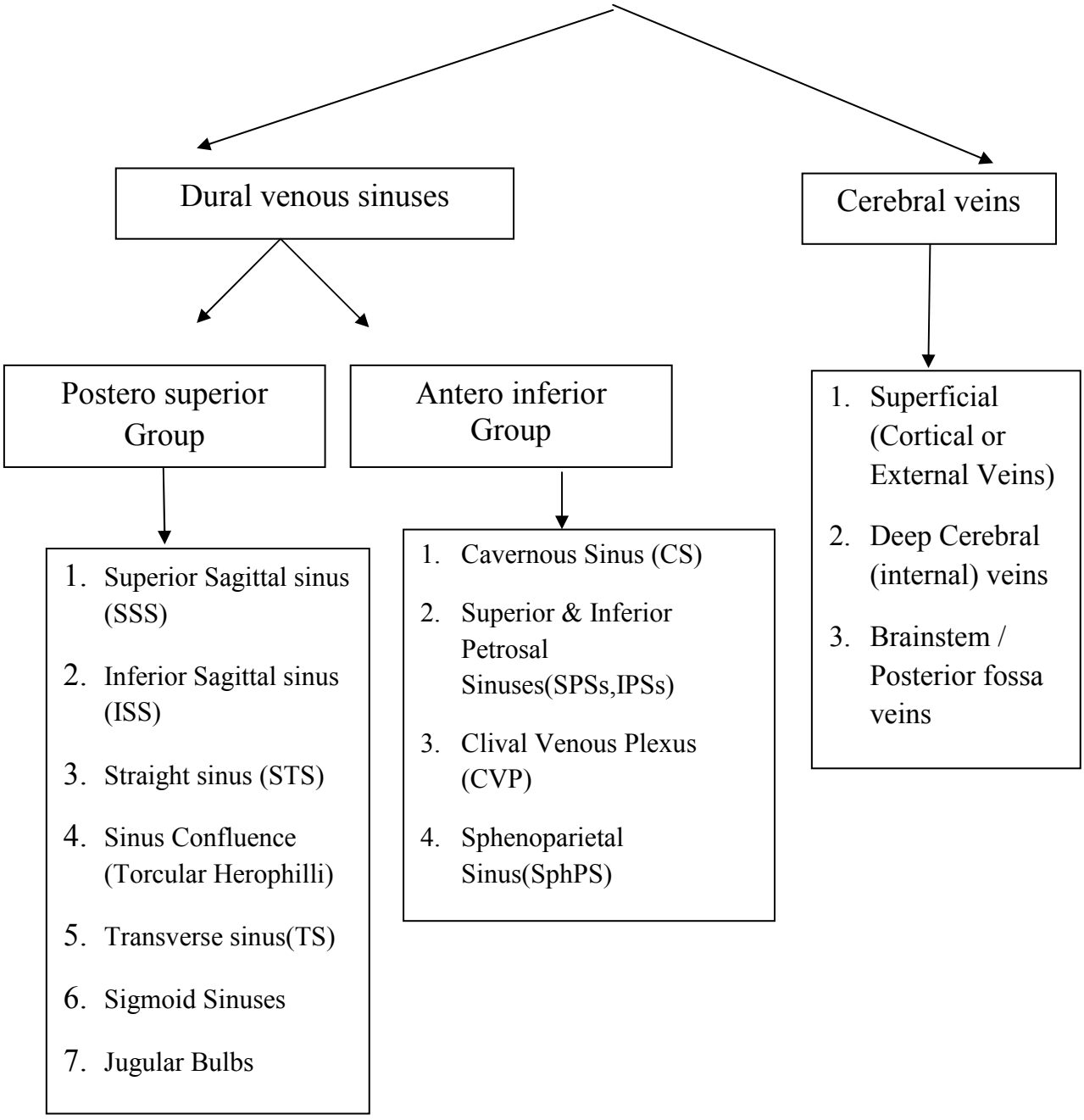
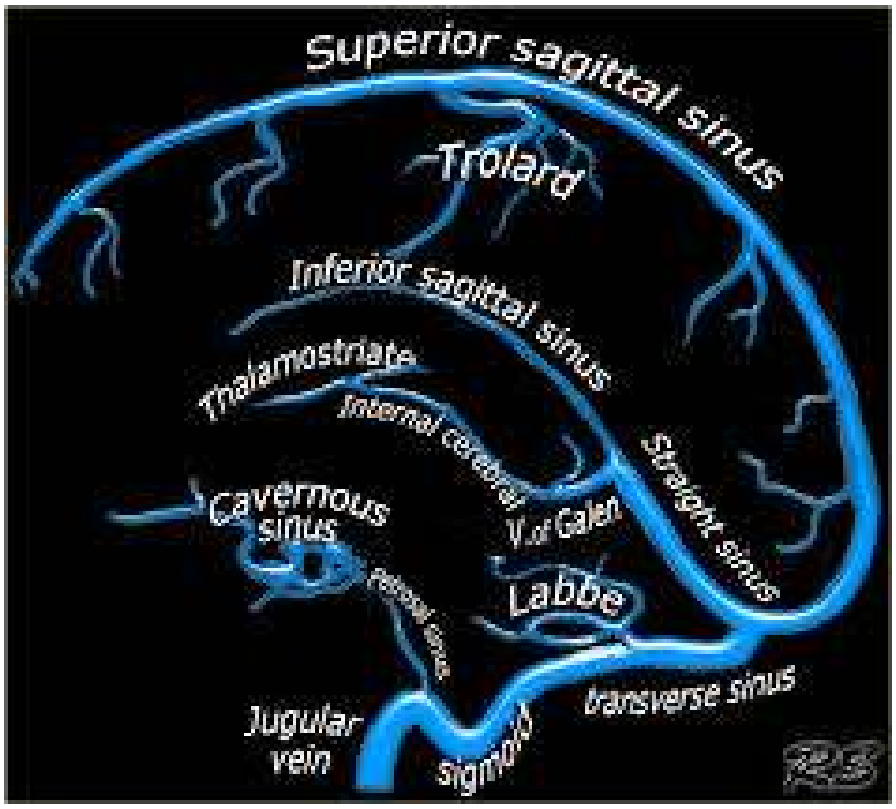


Figure – Schematic diagrams shows Superior sagittal sinus, straight sinus, sigmoid and transverse sinuses. Formation of great cerebral vein of galen also depicted.



Inferior anastomotic vein of labbe and superior anastomotic vein of trolard are shown in the schematic diagram

Figure: – (a). Schematic diagram shows cortical veins and superior anastomotic vein of trolard. (b). Shows dural venous sinuses and deep veins

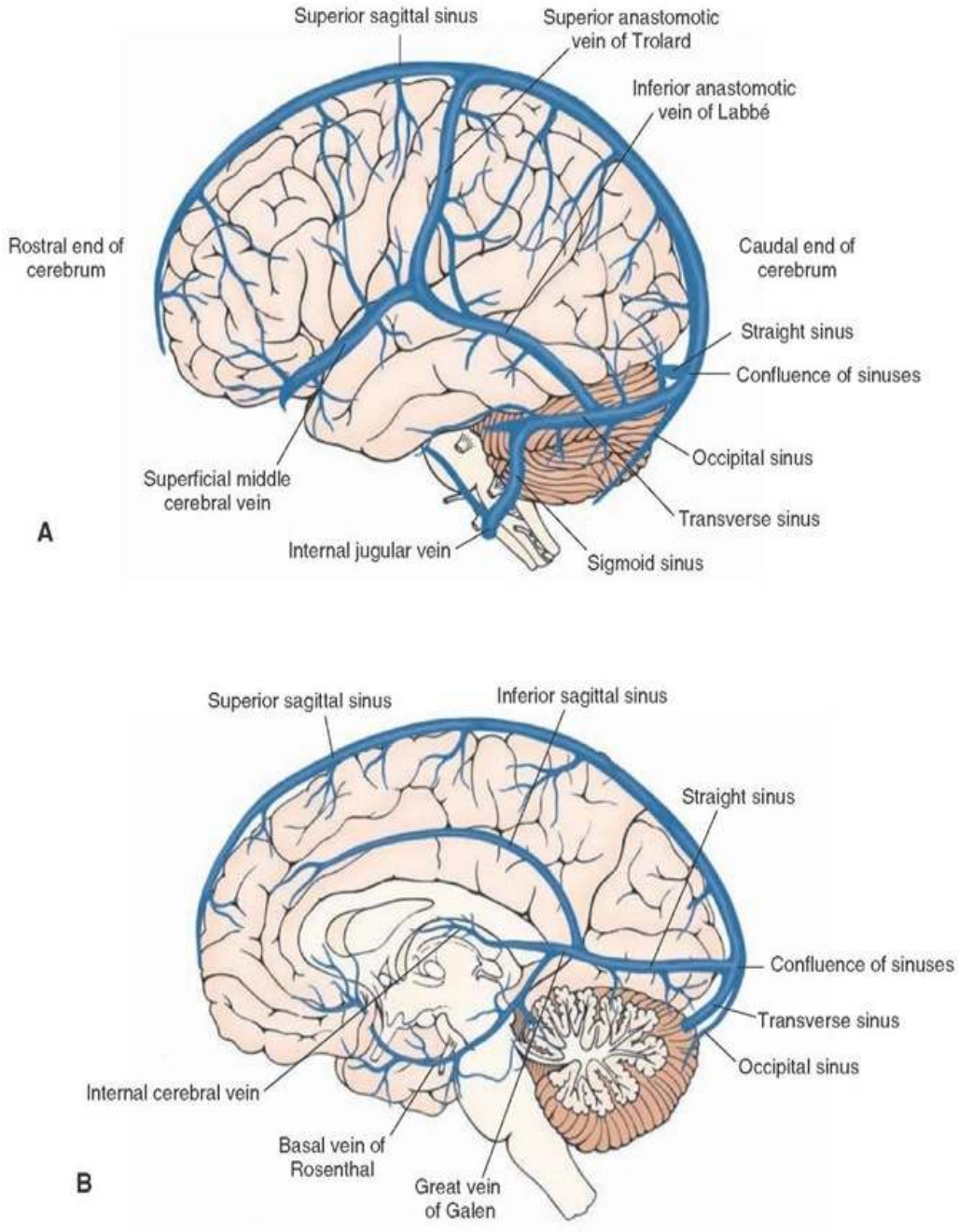
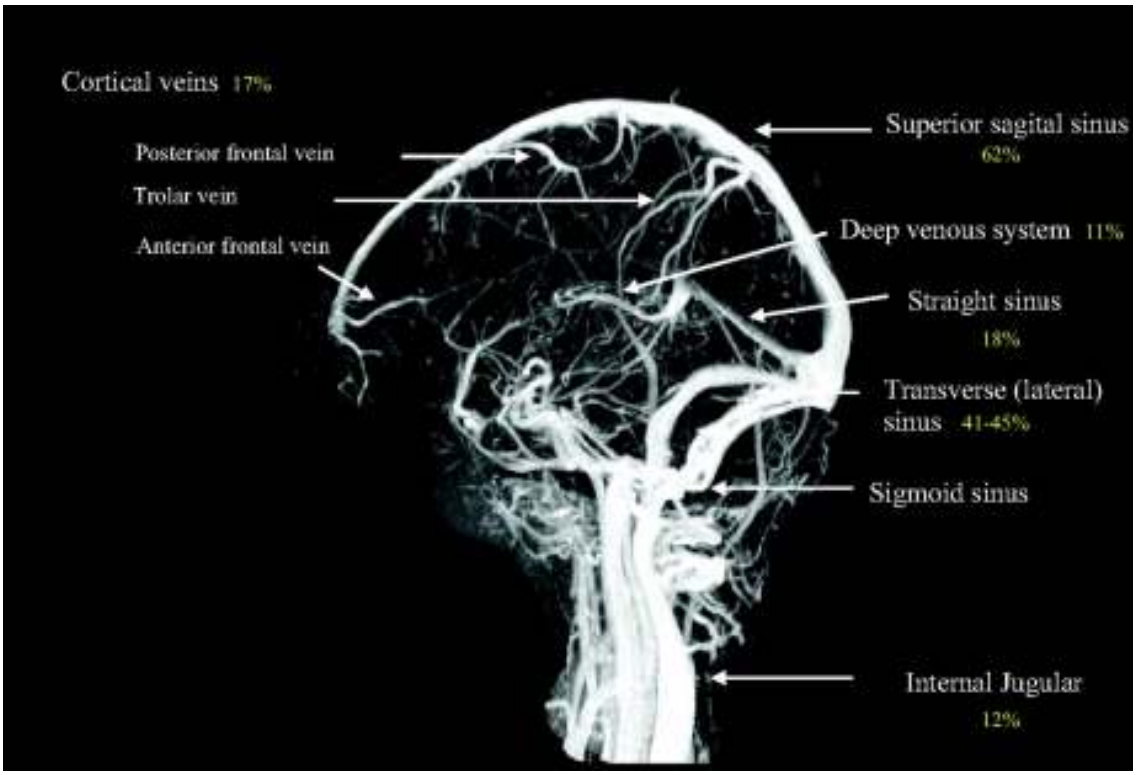


Figure:- MRV image showing superior sagittal sinus, transverse sigmoid and internal jugular vein, cortical veins and vein of trolard. Image also shows deep venous system.



CEREBRAL VENOUS THROMBOSIS

Cerebral venous thrombosis (CVT) has been identified to be the cause of 6% of maternal deaths. There seems to be heightened risk during the first 2 weeks of puerperium(12) particularly in young mothers and after caesarean section. Earlier CVT was presumed to be a rare as well as severe condition.

Pregnancy infact is highly demanding on the body metabolism. The cardiovascular changes and connective tissue changes prepare the pregnant women to undergo this challenging phase. Estrogen and other maternal hormones promote renin activity which causes retention of sodium and water eventually leading to an increase in plasma volume by around 6 weeks of gestation.

There is a paralleled physiologic hemodilutional anemia of pregnancy, 30% to 50% increase in cardiac output, stroke volume and heart rate commences by the 5^{th} week of gestation and peaks in the late second or third trimester. There is also concomitant rise in prostacyclin levels and redistribution of high flow in the low-resistance uteroplacental circulation and breasts and kidneys causes systemic vascular resistance (SVR) to drop by 5 weeks of gestation.

Pregnancy also induces remodeling of the heart and the systemic blood vessels. Arterial collagen and elastin content is decreased resulting in inability of the vessels to distend. Molecular factors responsible for it and its association with stroke are yet uncertain.

Hypercoaguability has a provocative role in the evolving of CVT during pregnancy and puerperium. Some of the prime events that further render a hypercoagulable state are dehydration due to antepartum as well as post partum hemorrhage, poor obstetric practices, trauma during instrumental delivery and inadequate intake of fluids inspite of breastfeeding(7).

Also venous stasis as a result of prolonged bed rest because of any complication arising during labour or following Caesarean section, worsen the prothrombotic state.

Based on the pattern of onset and how the thrombus has extended in the veins, CVT has a pleomorphic clinical presentation from headache, somnalence, coma, generalized seizures and neuropsychiatric symptoms. Focal neurologic deficits are also manifested(9). Nonseptic CVT commonly occurs in the superior sagittal sinus, whereas septic CVT involves cavernous and lateral sinus(10).

PATHOLOGY OF VENOUS THROMBUS

Thrombus occlusion of a dural sinus

Extention to involve bridging vein

Tributaries of cortical vein occlusion

Petechial perivascular hemorrhage and cortical venous infarction

Cerebral venous thrombosis in pregnancy and puerperium has more of a sudden onset yet better outcome compared to patients who develop thrombosis due to other pathological conditions(12).

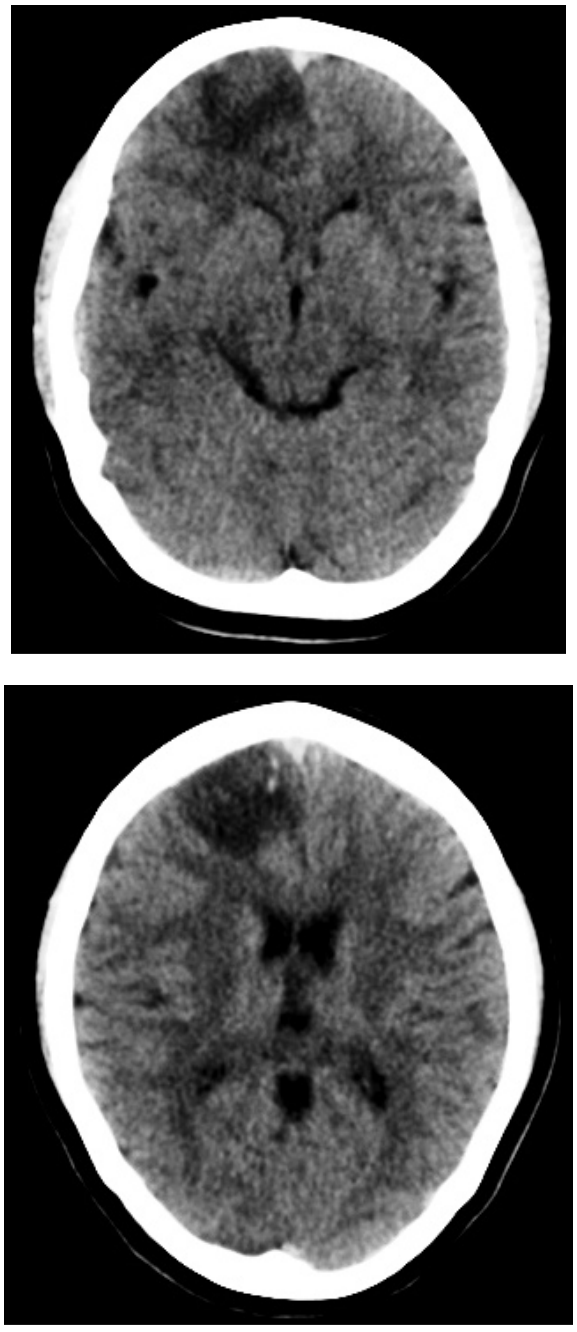


Figure – NE CT brain shows superior sagittal sinus thrombus with right frontal hemorrhagic infarct

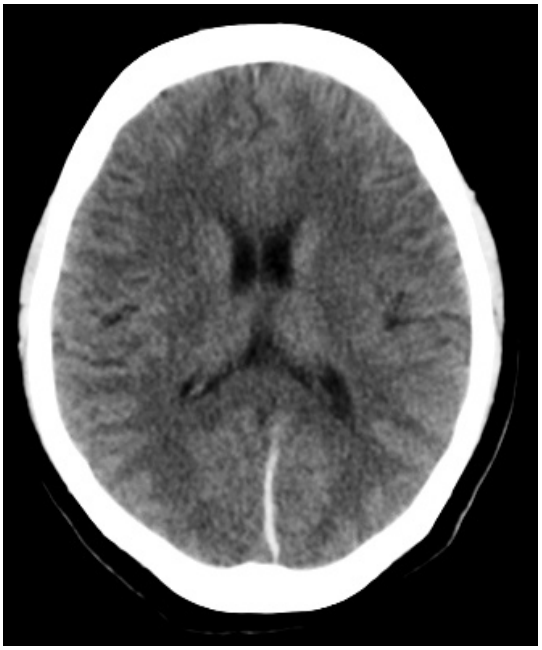


Figure – NE CT brain shows straight sinus thrombosis

IMAGING FEATURES OF CVT

CT imaging studies depict enhanced attenuation of the cerebral venous sinuses in which thrombosis has occurred. It may or may not be associated with infarction in the veins. Contrast-enhanced CT shows 'empty delta sign' which is a characteristic filling defect. Nevertheless it might take 7–10 days for the empty delta sign to appear on CT following the commencement of symptoms. In comparison to CT, MR imaging proves to be of better accuracy and sensitivity(11,13).

A credible sign of CVT is simultaneous high signal intensity of the venous sinuses with all routine sequences(14) (T1-weighted, T2-weighted, and FLAIR). High signal intensity on T1-weighted images with

a corresponding filling defect after gadolinium enhancement may develop within the first week after clinical onset.

Early detection can be done with MRI within7 days of clinical onset whereas CT imaging may take 7-10 days to show significant changes(11,14). An early feature is inadequate physiologic enhancement of venous sinuses which can be seen in both CT and MR images.

MR venography in addition to routine MR imaging, besides diagnosis help to understand the tributaries of the major cerebral veins and dural venous sinuses(13,14). Some of the other parenchymal signs of CVT are diffuse mass effect, localized sulcal effacement, and venous infarcts.

Venous infarcts are not restricted to the arterial zones and are often associated with hemorrhage at the gray-white matter interface.

Catheter angiography has more significance in the treatment of CVT rather than diagnostic purpose(17). It is used for administration of local thrombolytic agents and in retrieval of thrombus.

Anticoagulation is the recommended treatment modality though early thrombolysis has relatively better response even in the presence of hemorrhagic infarctions (15,17).

CVT has shown a commendable recovery(11) in cases where the occlusion is limited and transient with swift recanalisation or by formation of collateral circulation.

Neuropsychiatric manifestations and pseudo tumor cerebri have good prognosis whereas bilateral hemorrhagic infarctions and diffuse cerebral oedema present as an acute fulminant variant with comparatively grave outcome.

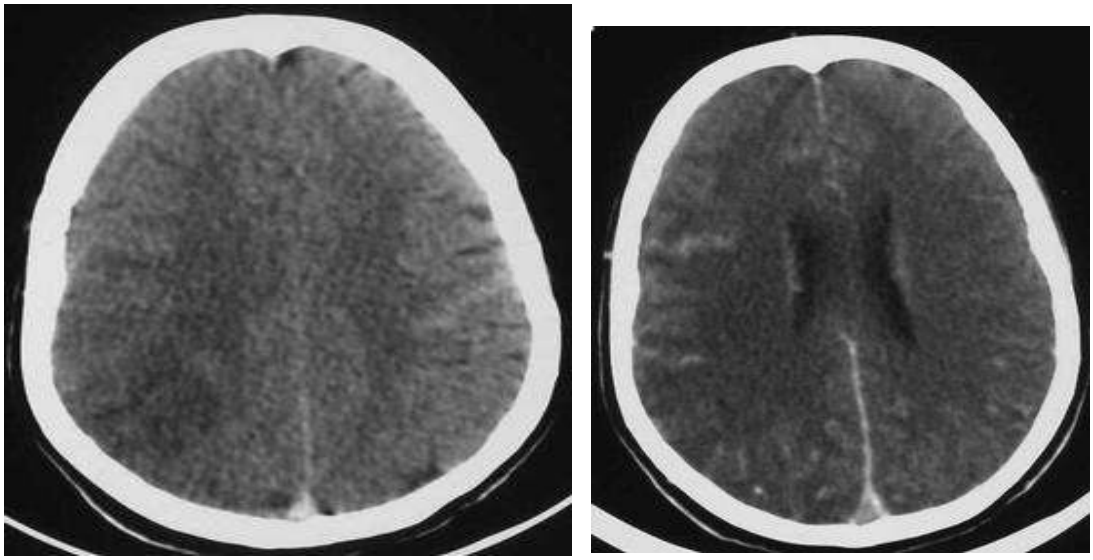
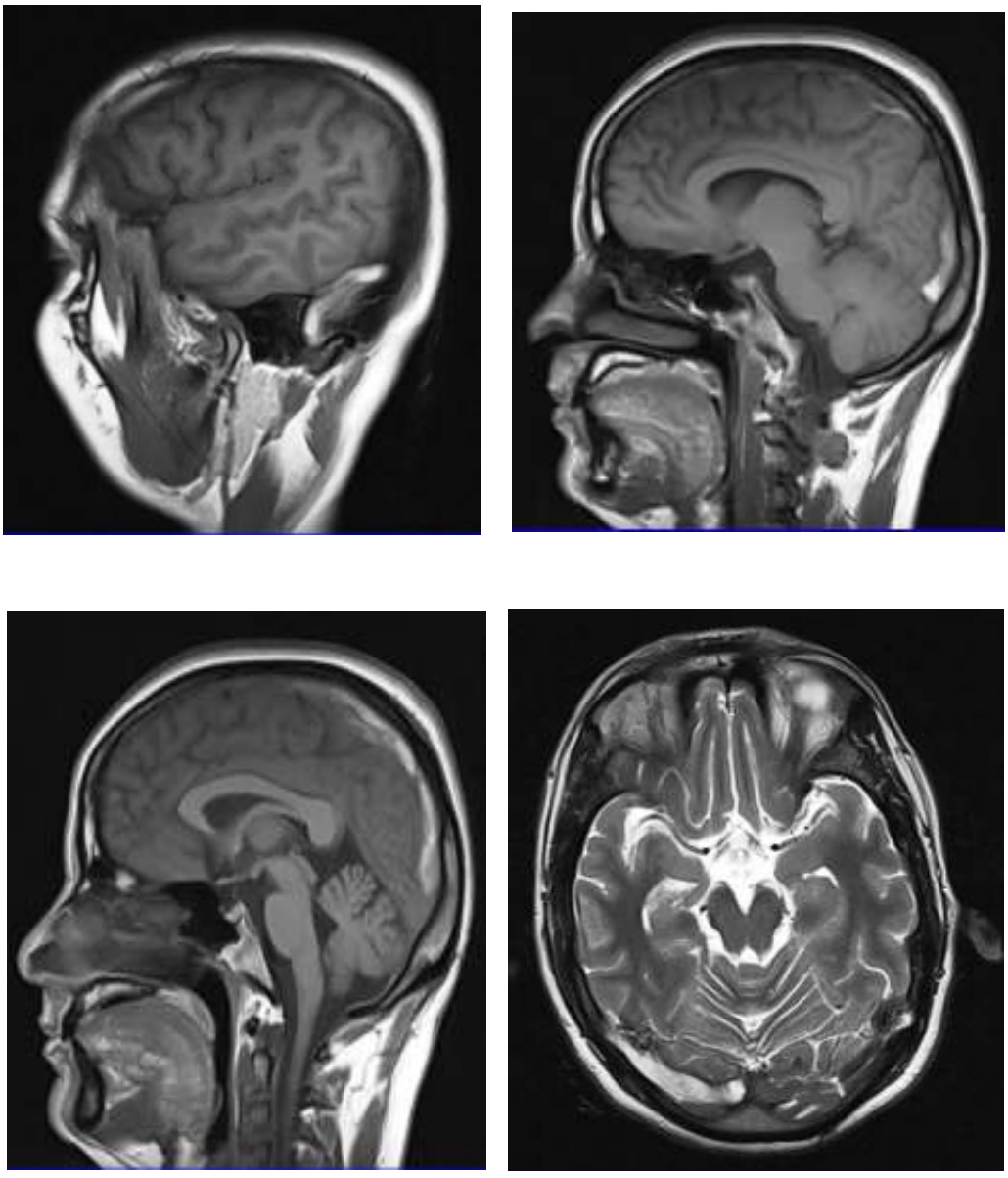


Figure –NE CT shows hyperdense superior sagittal sinus CECT shows empty delta sign

Figure- T1W sagittal T2 axial images shows hyper intense thrombus involving right sigmoid, transverse sinus, tarcular herophilli and superior sagittal sinus.



Differential diagnosis of CT and MR imaging of dural venous sinus thrombosis

CT

Normal variant

High – splitting of tentorium on CECT

Abnormal

SAH / SDH along tentorium and falx

MRI

T1 Weighted

Flow related enhancement (entry phenomenon)

Inplane flow / slow flow

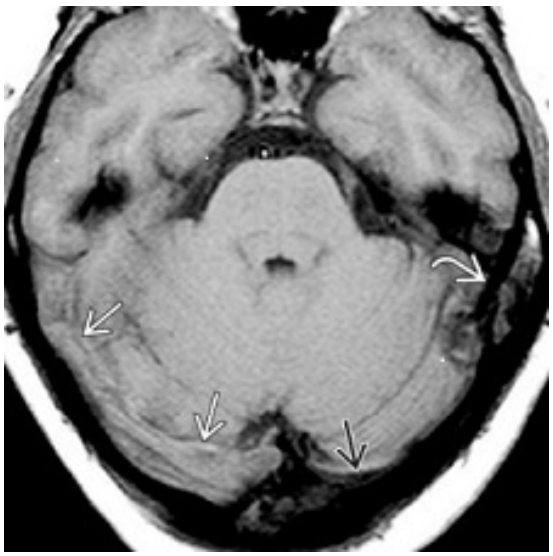
Post contrast flow compensated scan

T2 Weighted

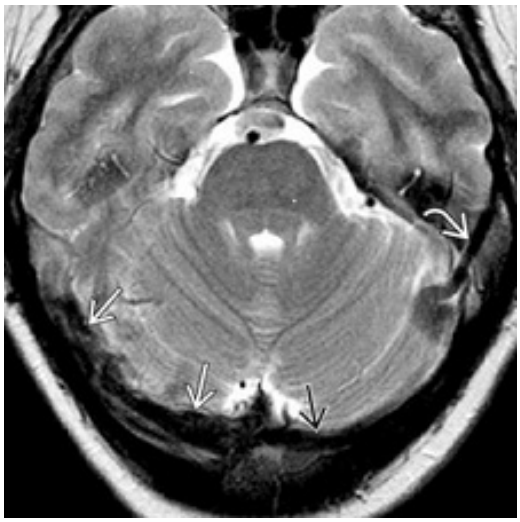
Cardiac pseudogating

Very slow inplane flow

ACUTE STAGE

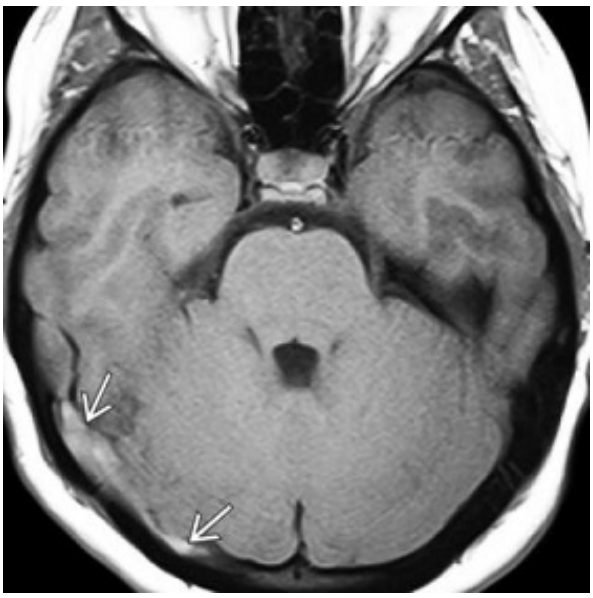


Axial T1WI in a patient shows an enlarged right TS that appears filled with isointense clot .Compare to the normal "flow void" in the left vein of Labbé and transverse sinus

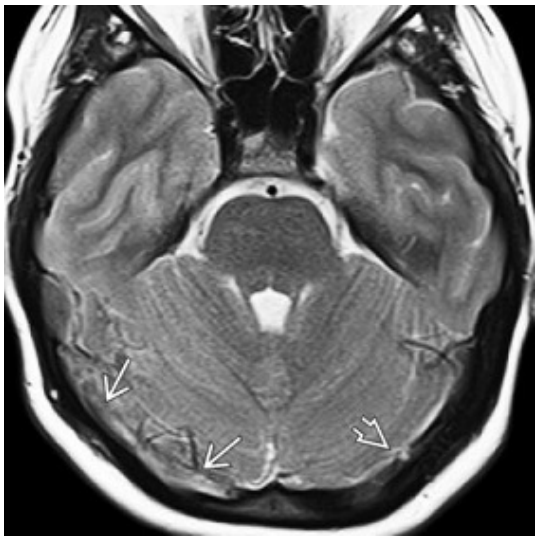


Axial T2WI in the same patient shows that the thrombosed right TS appears very hypointense and mimics the "flow voids" of the patent left TS and vein of Labbé

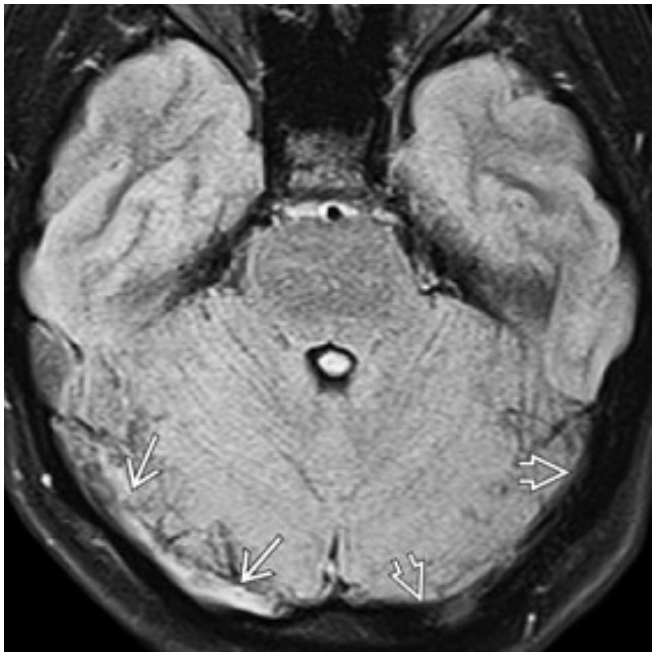
EARLY SUBACUTE STAGE



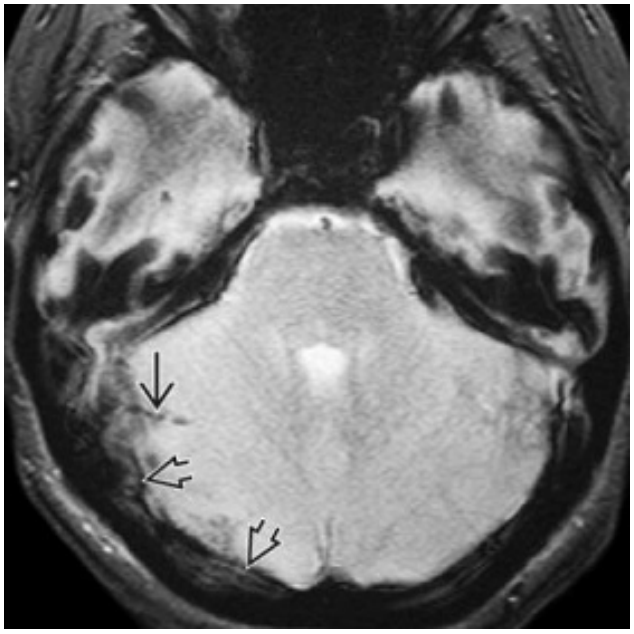
Early subacute DST in a 25-year-old man with several days of diarrhea and progressively worsening headache. NECT scan had demonstrated no definite abnormality. Axial T1WI shows mild hyperintensity in the right TS



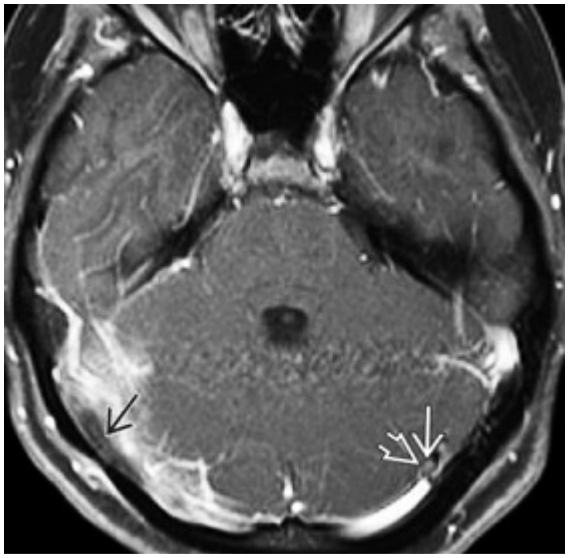
Axial T2WI shows that the thrombus in the right TS is beginning to appear mildly hyperintense, unlike the very hypointense clot seen on T2WI in acute DST. Note small T2 hyperintensity in the left TS



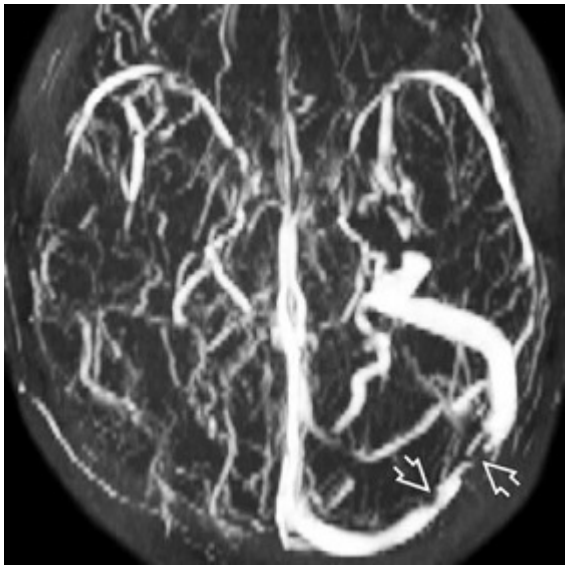
Axial FLAIR scan in the same patient shows that the right TS thrombus is mildly hyperintense. Contrast this with the normal "flow void" in the left TS



Axial T2* GRE in the same patient shows "blooming" thrombus in the right TS and tentorial venous tributaries

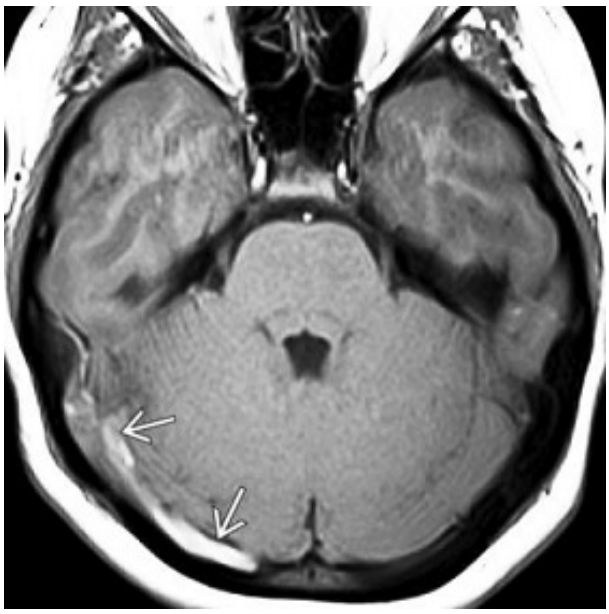


Axial T1 C+ FS scan shows the nonenhancing thrombus in the right TS surrounded by the intensely enhancing dura. The left TS shows an ovoid filling defect with CSF intensity containing a linear central enhancing vein. Findings are characteristic of an arachnoid granulation

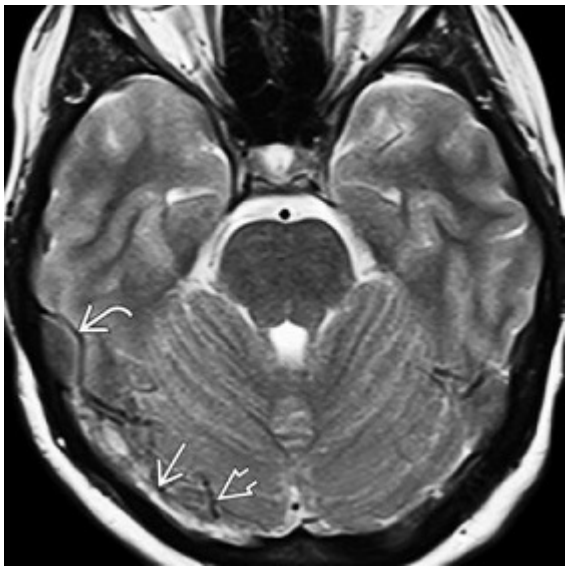


Axial MIP of 3D TOF MRV shows nonfilling of the right transverse and sigmoid sinuses. The 2 ovoid filling defects in the left TS are arachnoid granulations

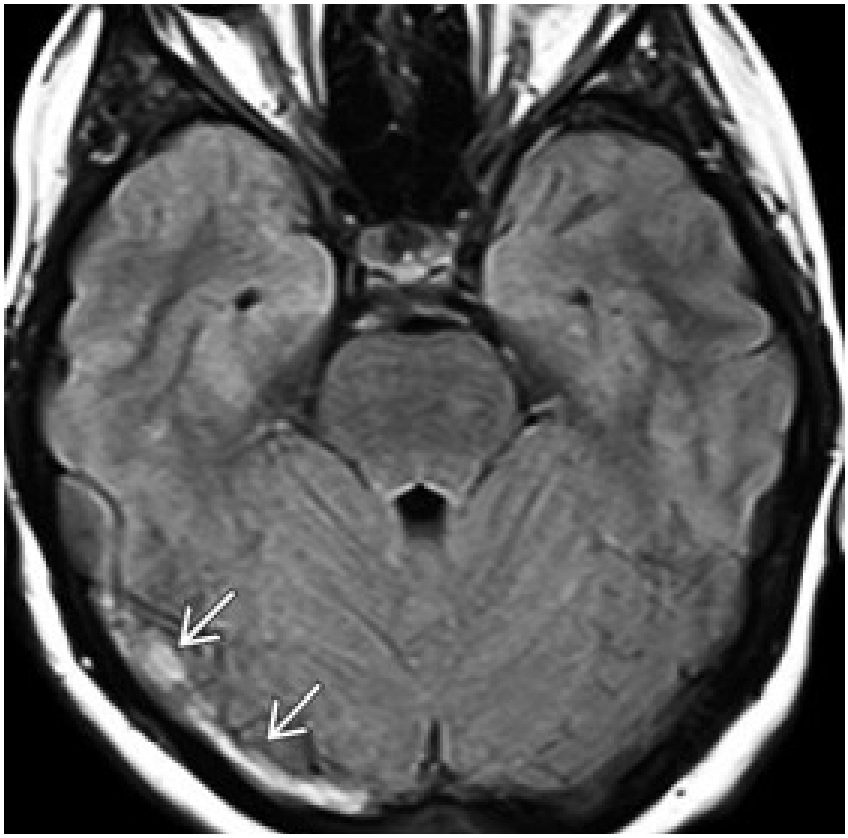
LATE SUBACUTE STAGE



Axial T1WI shows the striking hyperintensity of the late subacute clot in the right TS

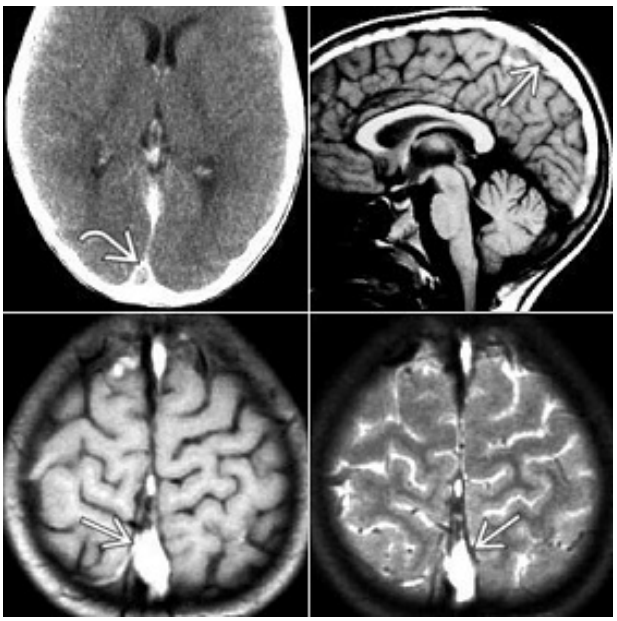


Axial T2WI shows the classic hyperintensity of late subacute thrombus in the right TS. Note normal "flow voids" in the patent adjacent vein of Labbé and tentorial tributary veins

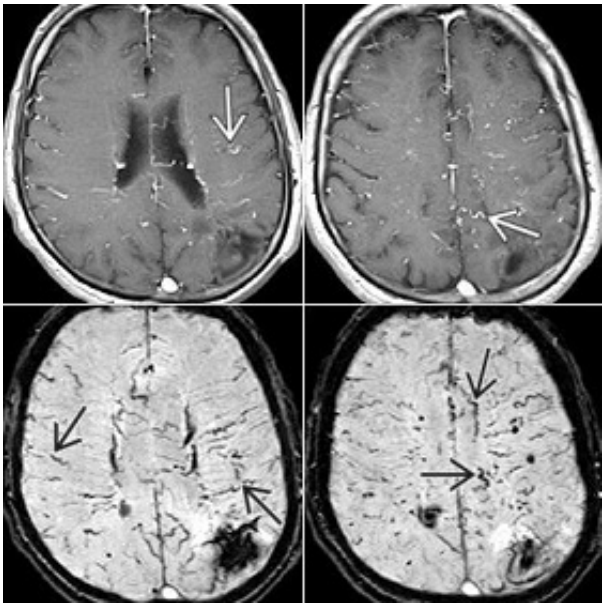


Axial FLAIR in the same patient shows the hyperintense late subacute thrombus. The adjacent cerebellum and posterior temporal lobe appear normal, without evidence of venous ischemia or infarction

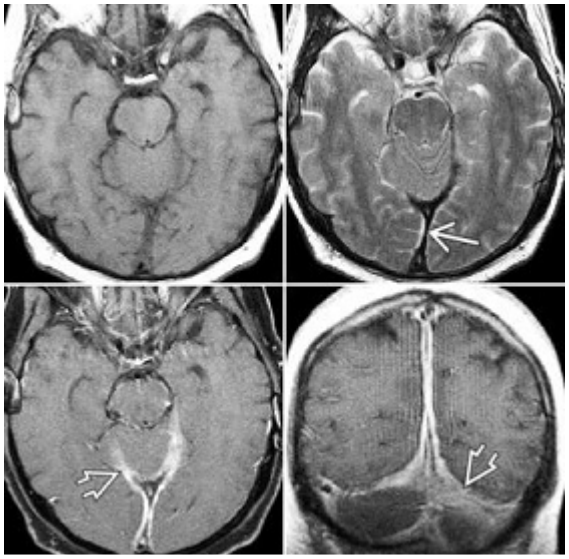
CHRONIC STAGE



Chronic SSS thrombosis shows "empty delta" sign and hyperintense thrombus



Chronic SSS occlusion shows prominent "squiggly" parenchymal veins on T1 C+ and "flow voids" on SWI



Chronic SSS thrombosis shows hypointense thick enhancing dura

TREATMENT

The management of CVT consists of symptomatic treatment and definitive treatment of the thrombus.

It Consists of

(i) Antiepileptics

(ii) Treatment of raised ICT

(iii) Antithrombotics

(iv) Treatment of primary cause.

Seizure is a poor prognostic factor. It results in secondary ischemia and brain damage. So control of seizures using antiepileptics both oral and parenteral remains one of the mainstay in CVT treatment.

CVT results in venous congestion, parenchymal edema, hemorrhage and infarcts. These results in raised ICT. This can result in transtentorial herniation of brain parenchyma and brainstem compression leading to sudden death. So prompt treatment of raised ICT prevents much of the mortality associated with CVT.

Treatment consists of medical and surgical methods. Medical management includes IV mannitol. Surgical treatment includes burr hole decompression of cranium, hemicraniectomy, surgical debridement. ***(Jam stom et al)***

Heparin remains the first line treatment of CVT ***(Marie Germaine et al)*** Recent studies shows its safety even in hemorrhagic lesions. It is followed by oral anticoagulants for atleast 12 months.

Local thrombolysis is reserved for those who do not respond well to the above treatment. Local urokinase infusion into the thrombosed sinus results in lysis of thrombus. It can be done through internal jugular or femoral vein routes. RtPA is a good alternative for urokinase. Complication include increased intracranial bleed and pelvic bleed from the puncture site.

Mechanical disruption of clot can be done using various mechanical devices. In case of deep vein thrombosis and cerebellar infarction ventricular drainage is used to relieve hydrocephalus.

HEMORRHAGE

Hemorrhage can be intraparenchymal or in subarachnoid space.

ICH

ICHs are most frequently due to etiologies also seen in nonpregnant patients. Chronic hypertension, pregnancy-induced hypertension, preeclampsia, or eclampsia frequently contributes to ICH during pregnancy and the puerperium.[23]

Pathology shows fibrinoid necrosis of small penetrating vessels, as in typical hypertensive hemorrhage. Arteriovenous malformations (AVMs) and aneurysms are more likely to rupture during pregnancy and the puerperium.

They are responsible for many ICHs and most subarachnoid hemorrhages (SAHs) during pregnancy and the postpartum period. AVMs usually bleed from the venous side, and can rupture at any time during pregnancy, perhaps due to increased blood volume and venous blood pressure. Aneurysms usually bleed in the latter half of pregnancy, and are more likely to rupture in a hypertensive patient.

Intraparenchymal hemorrhage appearance in T1 and T2 images vary with the stage of hemoglobin breakdown.

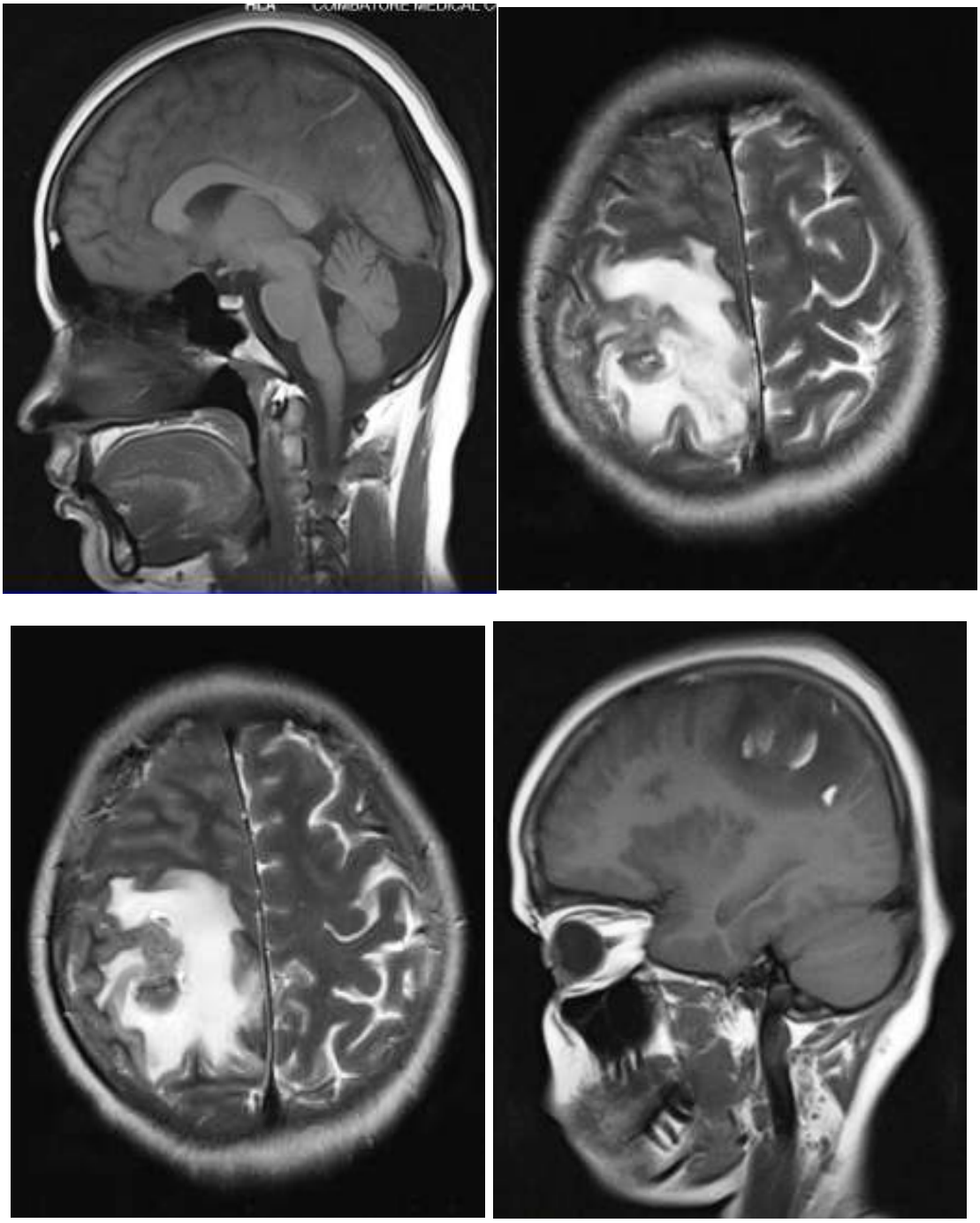


Figure – T1W sagittal T2 axial images shows superficial cortical vein thrombus with hemorrhagic infarctions

Five stages of evolution of hematoma are recognized using MRI characteristics on T1 & T2 weighted images.

They are

1. Hyperacute	-	<24 hrs
2. Acute	-	1 to 3 days
3. Early subacute	-	3 to 7 days
4. Late subacute	-	7 to 14 days
5. Chronic	-	> 14 days

In the hyperacute stage there is intracellular oxyhemoglobin. In the acute stage oxygen dissociates forming deoxyhemoglobin. During the subacute stage deoxyhemoglobin is oxidatively denatured to methemoglobin which is intracellular in early subacute and extracellular in late subacute due to RBC lysis. During the chronic phase heme is digested by the macrophages and converted into hemosiderin and ferritin.

The progression of hemoglobin catabolism within the hematoma starts in the periphery and extends into the center. So at a point of time the hematoma consists of hemoglobin stages of varying ages. But the hematoma should be aged taking into account the most advanced stage of hemoglobin catabolism.

The signal intensities of the hematoma depends on age of the hematoma.

The common pattern of signal intensities are

Sl.No	Stages	T1	T2
1.	Hyperacute (oxyhemoglobin)	Iso	Hyper
2.	Acute (Deoxyhemoglobin)	Iso /Hypo	Hypo
3.	Early Subacute (Intracellular methemoglobin)	Hyper	Hypo
4.	Late Subacute (Extracellular methemoglobin)	Hyper	Hyper
5.	Chronic (Ferritin, hemosiderin)	Hypo	Hypo

GRE sequences are more sensitive to hemorrhage ***(Luxia Liang et al)***. Since the 180° refocusing pulse of spin echo sequence is replaced by gradient pulses, the susceptibility is increased. Hemoglobin, because of its susceptibility effect causes signal loss of adjacent protons and appears hypointense.

FLAIR sequence is highly sensitive for subarachnoid hemorrhage ***(Rohit Baskhi et al)***. FLAIR is basically T2 image with suppression of free water protons. So the CSF is suppressed and hypointense in nature. This results in increased visibility of blood in subarachnoid space.

SUBARACHNOID HEMORRHAGE

Subarachnoid hemorrhage is a rare event in pregnancy. Intracranial aneurysmal rupture is the most common cause of SAH even in pregnancy and puerperium. The hemodynamic and hormonal effects of pregnancy enhance the risk of SAH owing to aneurysmal rupture, the occurrence of which is 1 in 10,000 patients. There is five times more increased incidence of subarachnoid hemorrhage in pregnant women, particularly in young primigravidas during the third trimester, than in nonpregnant women.

Management protocol of ruptured aneurysms remains the same as in patients who are not pregnant. In case of Un-ruptured aneurysms treatment is considered only if they are symptomatic or enlarging. Endovascular coil treatment of aneurysms has proved to be efficacious.

Occurrence of Subarachnoid hemorrhage without aneurysm is an extremely rare event[(18),] which is mainly due to pregnancy-induced hypertension. In pregnant women who died of eclampsia, leptomeningeal petechial hemorrhages were observed during autopsy. Rarely primary nonaneurysmal SAH owing to pregnancy-induced hypertension has been demonstrated by Computed tomographic imaging.

POSTERIOR REVERSIBLE ENCEPHALOPATHY SYNDROME.

PRES is a clinico radiological diagnosis.

Severity of clinical presentations vary among cases. MR and CT finding will also vary among the cases according to the severity.

Common symptoms of PRES are altered sensorium, seizures, severe headache, blurred vision, vomiting and focal neurological deficit.

COMPLICATIONS OF PRES

Cerebral ischemia

- Reported to occur in 10 to 25%.
- Non reversible damage of brain is associated with poor prognosis.

Cerebral herniation

- Severe edema in cerebellum and brainstern region will rarely cause transtentorial cerebral herniation.

Cerebral hemorrhage

- Uncommon complication in PRES occurs in 5 to 15% patient.
- Parenchymal hematoma, subarachnoid hemorrhage and intra parenchymal small focal hemorrhages (less than 5mm in diameter).

- Cerebral hemorrhage in patients with anticoagulant treatment.

DIFFERENTIAL DIAGNOSIS PRES

Posterior circulation stroke

- Differentiated by diffusion weighted images (DWI) which shows diffusion restriction in infarction cases, but not in the cases of PRES.

Reversible cerebral vasoconstriction syndrome

- There will be atleast two focal narrowings per artery on two different cerebral arteries at magnetic resonance angiography (MRA).
- There will be about 10% of overlap between posterior reversible encephalopathy and reversible cerebral vasoconstriction syndrome.

Primary CNS vasculitis

- In cases of vasculitis CSF analysis will be abnormal.
- Varying stages of multiple cerebral infarcts will be seen in vasculitis.

Encephalitis

- Differentiation between encephalitis and PRES will be by the distinct clinical history of PRES.

PROGNOSIS

- PRES is mostly reversible condition
- Clinically resolve after 3 to 8 days – MRI findings will take longer time to resolve.[23,24]
- Ideal time for repeat MRI study in case of PRES is 7 to 10 days.[25]

RADIOLOGICAL DIAGNOSIS OF PRES

There are usually four types of radiological presentation of PRES

1. **Holohemispheric watershed pattern.**

   Vasogenic edema in parieto occipital and frontal lobe white matter. Temporal involvement is rare in this type presentation.

2. **Superior frontal sulcus involvement pattern**

   Mainly edema occurs in superior frontal sulcus of frontal lobes.

3. **Predominant parieto occipital involvement**

   Mild to severe edema in posterior most parts of parietal and occipital lobe.

4. **Asymmetric presentation of primary pattern**

   There will not be any edema in parietal or occipital lobes.

   Common involvement in frontal lobe.

   Unilateral presentation can also occur.

CT AND MRI IMAGING IN PRES

- CT findings are usually normal. If there are specific hypodense areas in particular distribution, it suggests PRES.
- MRI is an important investigations for PRES diagnosis. FLAIR, T2w and PD will show high signal which indicates edema.
- FLAIR useful in areas of sub cortical and cortical lesions.
- ADC will be increased.
- Few cases will show enhancement also (50%)
- For PRES diagnosis MRI is superior one.

PATHOPHYSIOLOGY OF PRES

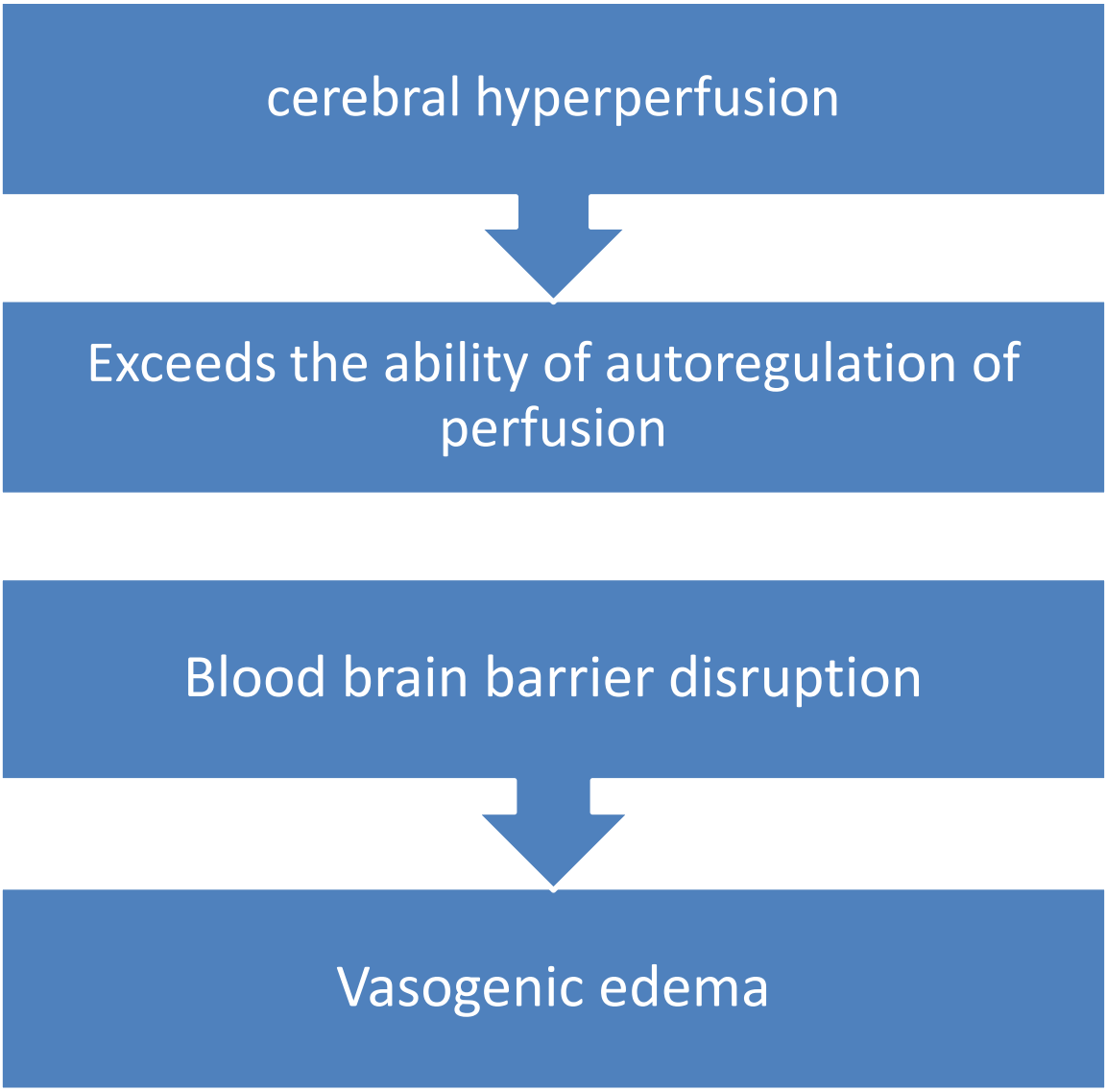
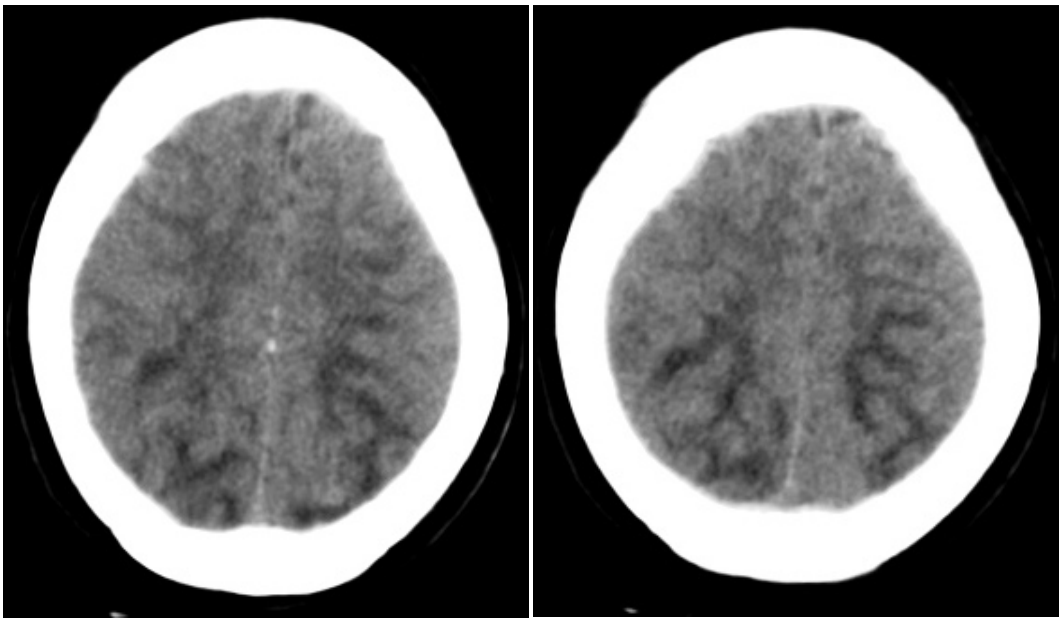


Figure - NECT brain shows bilateral parietooccipital edema suggestive of posterior reversible encephalopathy syndrome



POSTPARTUM CEREBRAL ANGIOPATHY

Postpartum Cerebral Angiopathy is a rare disorder that affects otherwise normal women who have undergone an uneventful pregnancy as well as delivery. These normotensive postpartum women present with severe headache, seizures, and focal neurologic deficits within 1–4 weeks of delivery.

Two distinctive variants of PCA have been identified by studies. Idiopathic PCA also known as Call-Fleming postpartum angiopathy is a reversible nonrelapsing angiopathy. Iatrogenic PCA occurs anytime during the puerperium either due to use of medications like bromocriptine used to suppress lactation ergot alkaloids used to control postpartum hemorrhage or due to use of sympathomimetics and nasal decongestants for respiratory tract infection.

PCA is a reversible type of clinic - radiological syndrome. Symptoms include sudden onset of headache associated with nausea. Focal areas of neurological deficit occur due to vasoconstriction in cerebral blood vessels.

Definitive diagnosis for this condition is by cerebral angiography. Which will shows multiple areas of focal and segmental narrowing,

mainly in large sized and medium sized vessels. The imaging findings of cerebral angiopathy are identical to the features of vasculitis.

Focal narrowing of vessels will get corrected within 1 to 3 months. However severity of cerebral angiopathy is associated with infarction and hemorrhage. There is a significant risk of morbidity and mortality associated with PCA.

Two thirds of PCA occurs within the first 7 days of delivery.

DIAGNOSIS

- Definitive diagnosis with angiography
    - String of beads like multiple segment narrowing.
- CT angio or MR angio are 80 % sensitive
    - T2/Flair hyper intensity in watershed areas
- Angiogram will be normal in early stages of disease, that is within 4 – 5 days. But a second angiogram few days later may reveal positive findings.

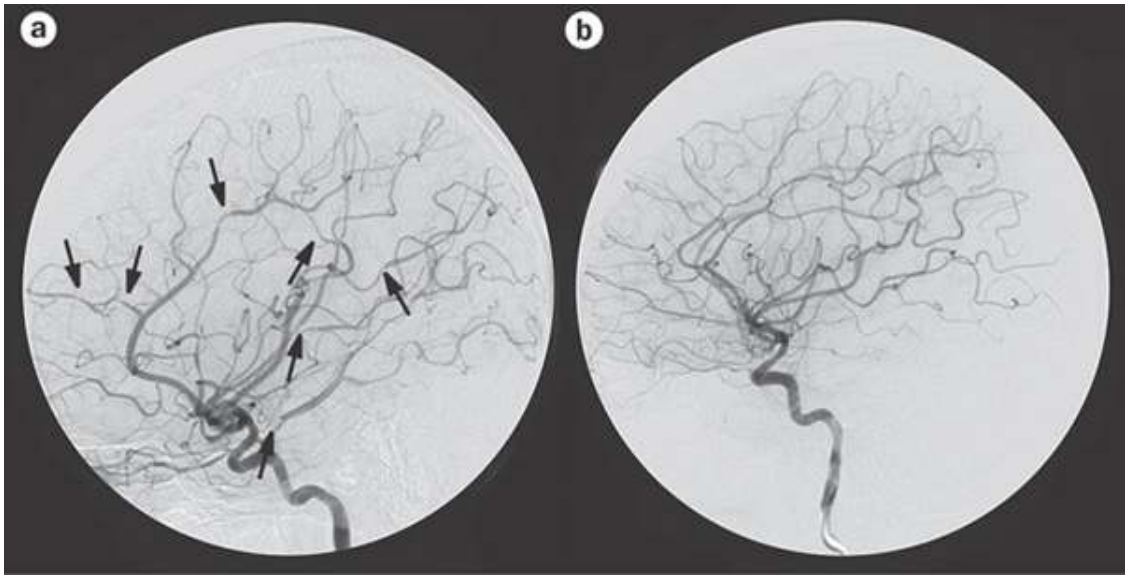
The diagnosis of PCA should be considered in normotensive postpartum women presenting with intracerebral hemorrhage. At imaging, there is intracerebral hemorrhage with local mass effect.

Patients with PCA may also develop reversible high T2 signal abnormalities anywhere in the brain cortex or white matter.

Angiogram shows reversible multifocal stenoses and post stenotic dilatations in the medium and small sized cerebral arteries in the anterior circulation. Though the angiographic findings may be identical with vasculitis arising as an inflammatory process due to infection or some drugs, the features are more consistent with PCA. However, this is in contrast to eclampsia, which affects large and medium-sized arteries in the posterior circulation.

Transcranial Doppler ultra-sonography (US) reveals high flow velocities in the major cerebral arteries which is indicative of vasospam. This imaging modality is made use of to infer the potential benefits of treatment and thereby confirming reversibility of the vasospasm.

Treatment of PCA with corticosteroids such as intravenous methylprednisolone, calcium channel blockers such as nimodipine and hyperosmolar hypervolemic infusions have been suggested. Most of the patients recover without any permanent neurological deficits. Intracranial balloon angioplasty is rarely needed.



Angiography image shows multiple focal segmental cerebral arterial narrowing in a case of PCA

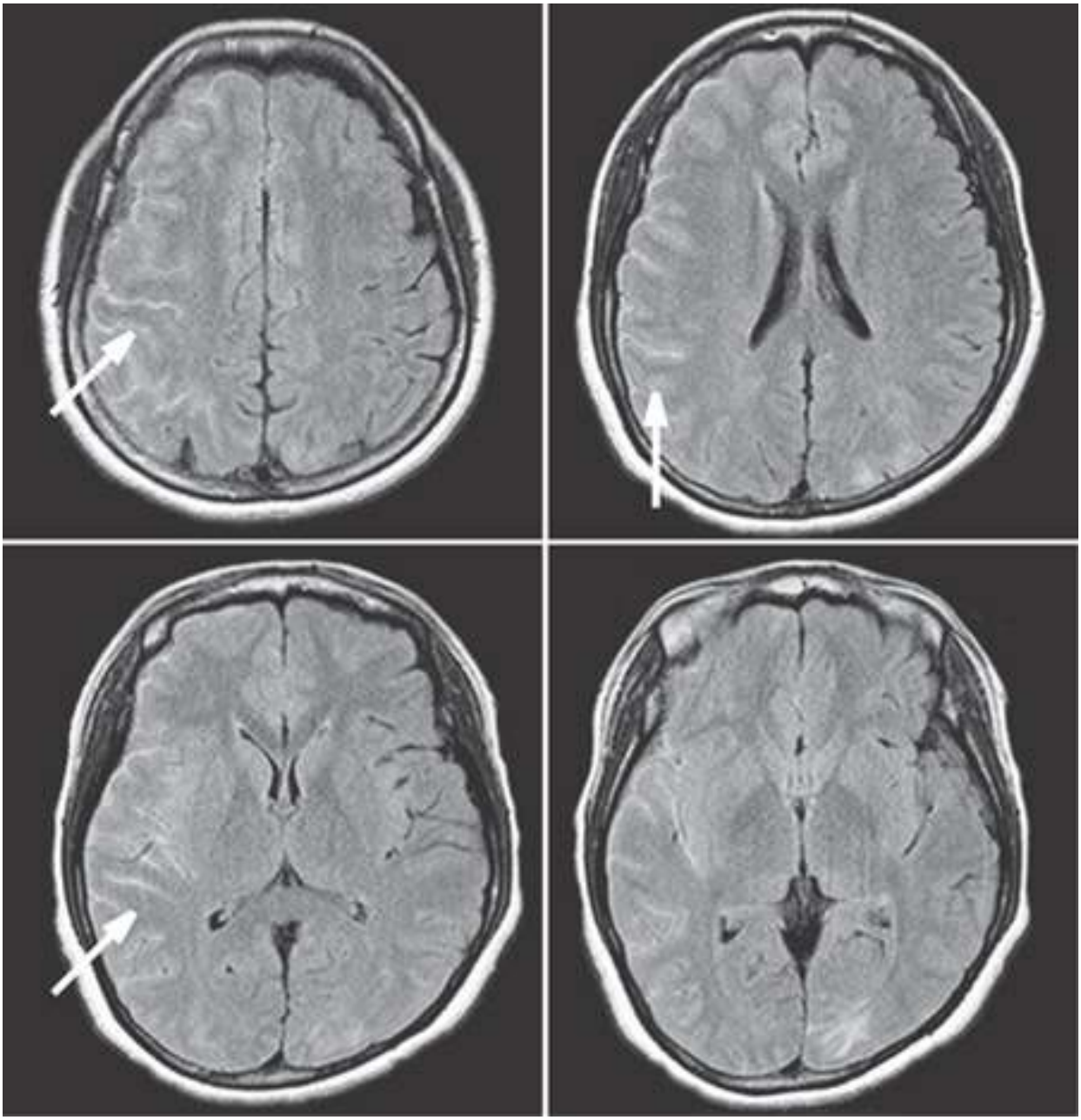


Figure – MRI image shows subarachnoid hemorrhage

in a case of PCA

PITUITARY APOPLEXY

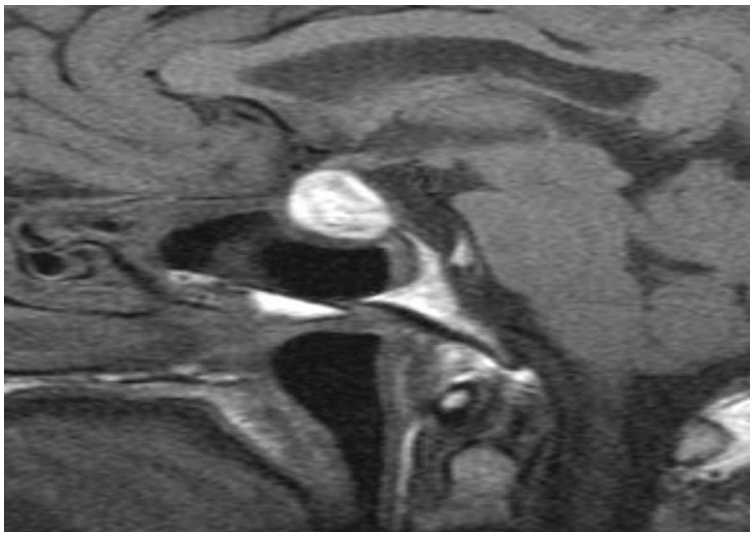
Pituitary apoplexy is defined as acute hemorrhagic infarction in an existing pituitary adenoma or otherwise physiologically enlarging pituitary gland. There is a predominant insufficiency of the adrenal hormones. Pituitary apoplexy is a rare condition during pregnancy which manifests with clinical features that include severe headache, vomiting, and visual disturbances including visual field defects and restricted eye movements.

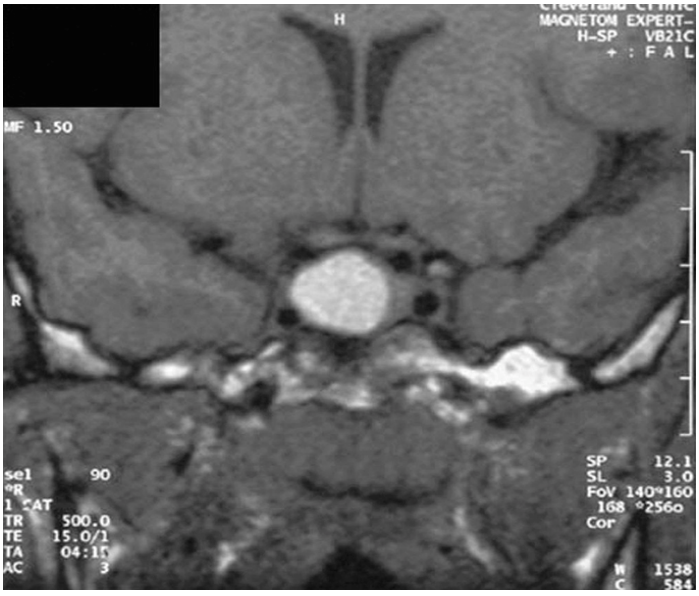
Sometimes it may be associated with meningeal irritation if there is minimal SAH. In acute hypopitutarism patient complains of dizziness or altered mental status probably due to hemodynamic instability. The neurologic examination, along with ophthalmologic fundoscopic examination and visual-field screening, is more often normal.

Magnetic resonance imaging (MRI), consisting of a T_2-weighted image in the axial plane and a T_1-weighted image in the mid sagittal plane, reveals an intrasellar mass with suprasellar extension and fluid levels denote recent bleeding into the macroadenoma. These findings are the hallmark of pituitary-tumor apoplexy. Intrasellar hemorrhage may not be visualized on imaging in some patients with pituitary apoplexy.

Supportive treatment with hormonal replacement and bromocriptine is more than sufficient. There is usually a spontaneous recovery after delivery. A follow up MRI shows that there is no sequelae. Trans-sphenoidal surgery is a safe procedure but not usually required.

T1 sagittal and coronal images shows enlarged pituitary with hyperintense signal suggestive of **macroadenoma with apoplexy**





SHEEHAN SYNDROME

Sheehan syndrome also called Simmond syndrome is due to ischemic necrosis of the pituitary gland. It is a clinical state of panhypo-pituitarism that ensues following pituitary infarction.

The anterior pituitary gland has a distinctive circulation in the form of a low pressure portal venous system. During pregnancy there is a diffuse and nodular enlargement of the lactotrophs of the anterior pituitary under the influence of estrogen secreted by the placenta.

Nevertheless, there is no compensatory increase in the blood supply. So the anterior pituitary gland is much vulnerable to a hemorrhagic episode attributing to ischemia and necrosis. Anti-pituitary antibodies have been speculated but diagnostic limitations have made it a less understood aspect. The posterior pituitary is mostly not involved due to its direct blood supply.

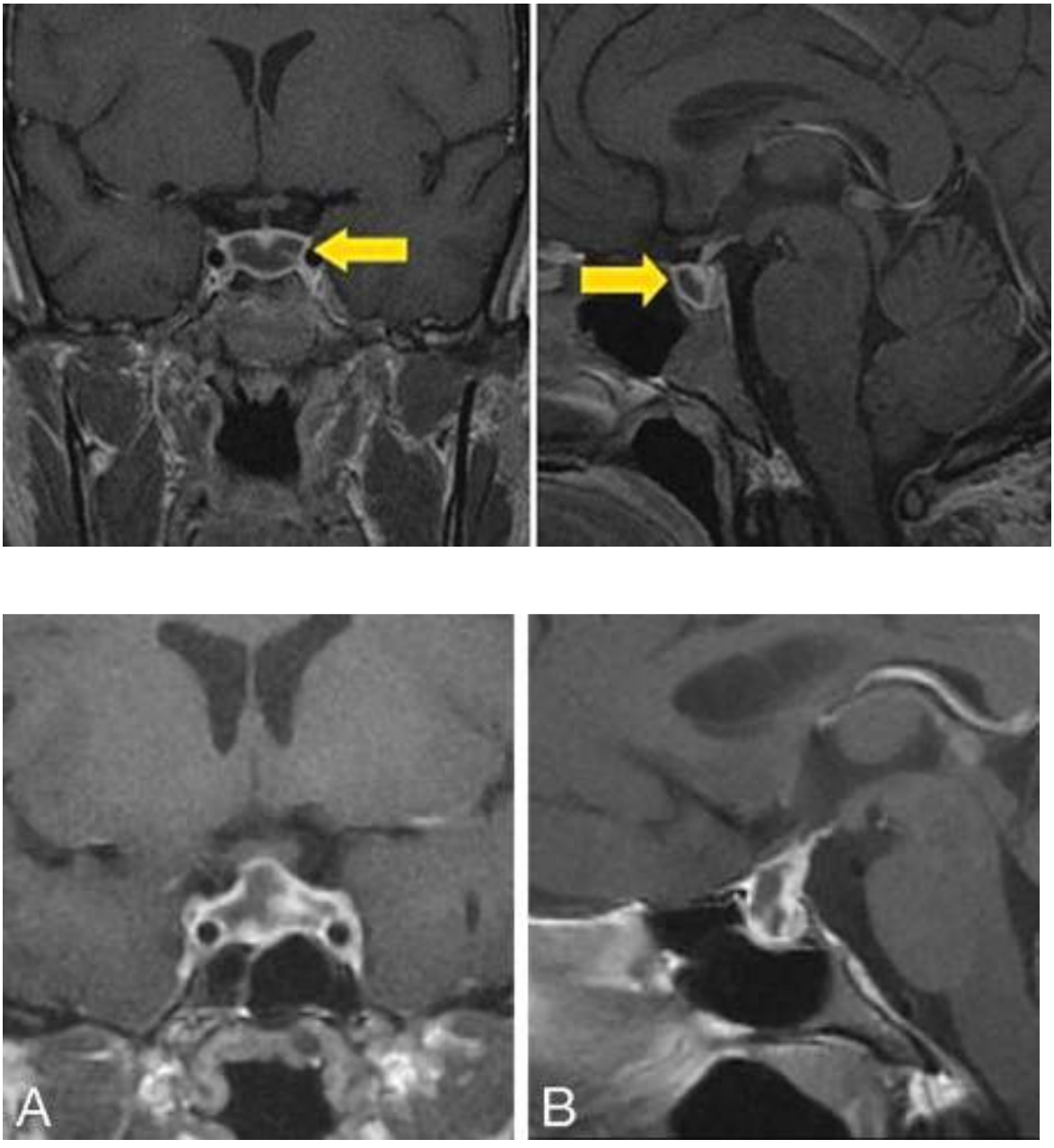
Most often it occurs due to post partum hemorrhage which results in hypotension and tachycardia. In the setting of an obstetric hemorrhage, if hypotension and tachycardia persist inspite of adequate treatment of the inciting event, pituitary infarction should be suspected. In the early phase, panhypopituitarism may precipitate hypoglycemia and failure of lactation.

Due to deficiency of pituitary hormones, depending on the extent of tissue detruction patients eventually present with chronic fatigue, dizziness, postural hypotension, cold intolerance, hypopigmentation, myxedema, loss of pubic and axillary hair, decreased libido, breast atrophy, and amenorrhea .

Some women with Sheehan syndrome might be relatively asymptomatic, and the diagnosis may be made coincidentally.

On imaging, partial or complete empty sella is visualized.

Figure - Coronal sagittal images of pituitary with contrast enhancement shows necrotic areas suggestive of pituitary necrosis.



LYMPHOCYSTIC ADENOHYPOPHYSITIS

Lymphocystic adenohypophysitis is a rare inflammatory disorder of the anterior lobe of the pituitary gland that may affect young women in the peripartum. The disorder has been reported only rarely in males, with a female to male ratio of approximately 10:1.

This disorder is considered an inflammatory autoimmune disease and has been associated with other autoimmune diseases such as autoimmune thyroiditis and pernicious anemia.

At imaging, there is enlargement of the pituitary gland with suprasellar extension in 60% - 80% of patients.

In lymphocystic adenohypophysitis, the pituitary gland may have variable appearances on MR images. In most of the patients, there is early and homogeneous enhancement of the pituitary gland. Though, heterogenicity may also be visualized in hypertrophied pituitary gland. Hemorrhage has not been reported in lymphocystic adenohypophysitis, to our knowledge.

Thickening of the infundibulum and involvement of the neurohypophysis resulting in diabetes insipidus are reported in **15**% of patients. There are no imaging features that distinguish lymphocystic

adenohypophysitis from pituitary adenoma. After recovery, there is regression of the pituitary gland to normal or small size, resulting in the appearance of a partial or total empty sella.

CT

CT with Coronal sections and reconstructions in multiple planes are used to image the pituitary region reasonably well.

Lymphocytic hypophysitis will appears as a soft tissue mass with enhancement from pituitary and extending superiorly into the suprasellar region.

MRI

MRI, as in other pituitary lesions, is the better modality for assessing this condition.

- **T1**
    - Affected pituitary region will appear isointense with slight signal heterogenecity.
    - Absence of bright spot in posterior pituitary, which is a normal finding.

- **T1 C+ (Gd)**
    - Variable enhancement, which is usually a homogeneous enhancement.
    - Enhancement of duramater can be seen.
    - Thickening of infundibulum.
- **T2**
    - Hypointensity in parasellar region useful in differentiating apoplexy from a pituitary adenoma

Steroids have been advocated as the main line of treatment. Glucocorticoid replacement therapy is beneficial due to its a potent anti-inflammatory effect.

PITUITARY ADENOMA

Prolactinomas are the most common pituitary tumors occurring during pregnancy. Increased levels of estrogen promote the growth of prolactinoma. The pituitary gland shows profound hyperplasia during pregnancy.

Proliferative growth and glandular enlargement commences in the early weeks of pregnancy attaining a size of 1.2 cm in diameter in the immediate postpartum period. This hyperplasia along with a corresponding increase in number of lactotroph cells subsequently leads to a gradual increase in serum prolactin levels from 35ng/mL in the first trimester to 175ng/mL in the second trimester peaking up to 210ng/mL before delivery .

There is a concomitant surge in estrogen hormone during pregnancy which is believed to influence the mitotic activity of lactotrophs and induces prolactin synthesis.

Pituitary evaluation is a confounding task in pregnant women. This is mainly due to the progressive increase in prolactin level. For imaging studies, use of gadolinium contrast material is not recommended in pregnant women due to its teratogenic effects.

When there is a definitive need for imaging, high-resolution MR imaging sequences without contrast material can be done. Routine estimation of prolactin levels are insignificant and do not serve the purpose of clinical assessment. Tumor growth is monitored at regular intervals by clinical assessment and visual field testing. Precisely, if the pituitary height exceeds 12 the diagnosis of pituitary adenoma is made.

The stimulatory effects of pregnancy on prolactinomas was established in the early 1970s especially in women who had prior treatment with ovulation-inducing agents or bromocriptine, Some patients recovered spontaneously following parturition while others required corrective surgeries for visual defects. Medications such as bromocriptine or cabergoline are considered the ideal treatment for prolactin-secreting microadenoma and adenomas limited to the zone of the sella.

Transsphenoidal surgery is mandatory in uncontrollable suprasellar macroadenomas. Even adenomas secreting adrenocorticotropic hormone should invariably be surgically excised. Surgery and medication are not beneficial in growth hormone–secreting adenomas and nonfunctioning adenomas. In patients with thyroid-stimulating hormone secreting tumors, antithyroid drugs can bring about a regression of hyperthyroidism state.

PRIMARY INTRACRANIAL TUMORS

Pregnancy augments the growth of meningioma. About 70% of meningiomas have progesterone receptors and 30% have estrogen receptors. Serial imaging reveal that size of the meningiomas deteriorate after delivery.

Surveillance propounds the stimulative effects of progesterone on tumor growth. Besides, tumors like hemangioblastoma and vestibular schwannoma has been reportedly seen to show enhanced growth during the course of pregnancy. However, pregnancy has no conspicuous effects on glioma.

ECLAMPTIC ENCEPHALOPATHY

Eclampsia is a critical condition seen in 5% of pregnancies and is responsible for 10% of the obstetric deaths. Eclampsia clinically presents with tonic-clonic seizures or coma in antenatal women particularly young mothers who already have developed pregnancy-induced hypertension.

It is not related to pre-existing organic lesions of the brain. Exact etiology is yet unknown. Imminent symptoms are headache, altered mental status, cortical blindness, and seizures. Eclampsia / PIH is one of the important cause for posterior reversible encephalopathy syndrome.

Owing to cytotoxic effects on the vascular endothelium there is increased permeability and vasogenic edema. Moreover, variable degrees of vasospasm and vasodilatation are due to acute variations in blood pressure. Any imbalance in the cerebral autoregulation predisposes to disruption of the blood-brain barrier in the posterior circulation.

Eclampsia and hypertensive encephalopathy are seen to have analogous imaging findings. CT demonstrates transitory posterior areas of patchy low attenuation.

MR imaging is has proven to be better than CT in patients with eclamptic encephalopathy. Lesions are characterized by low signal intensity on T1-weighted images and high signal intensity on T2-weighted images in the posterior cortex and subcortical white matter. Lesions conventionally show no diffusion restriction.

Diffusion-weighted imaging helps to differentiate between reversible vasogenic edema from cytotoxic edema of complete infarction. Sometimes there is involvement of the basal ganglia and brainstem. Catheter angiography classically shows vasospasm in the medium and large cerebral arteries, notably involves the basilar artery.

Prompt treatment of eclampsia with magnesium sulfate to prevent further convulsions in eclampsia is quiet essential. Antihypertensive

therapy with hydralazine or labetalol also plays an important role. Appropriate management of the hemodynamic instability will revert the adverse effects of hypoperfusion.

AMNIOTIC FLUID EMBOLISM

Amniotic fluid embolism is a rare cause of stroke. Amniotic fluid embolism develops when amniotic fluid trapped in uterine veins is forced into the maternal circulation. Presentation is typically an acute hemodynamic collapse, the exact etiology of which is not clearly understood. Consumptive coagulopathy develops in a majority of patients.

Focal neurologic deficits can be seen due to cerebral hypoperfusion or due to direct hemorrhage or thrombosis. Given the nature and severity of the syndrome, focal neurologic deficits are not usually seen in isolation. Amniotic fluid can travel to the brain through a patent foramen ovale as well, although this is quite rare.

INTRACRANIAL METASTASIS

Breast cancer and choriocarcinoma in pregnancy are notorious for manifestion as cerebral metastasis. Pregnant women who are in the age group of 32-38 are susceptible to carcinoma of breast, the incidence being recorded to be 1 in 3,000 pregnancies.

Unfortunately, during pregnancy and lactation the breasts tend to be firm, engorged and tender. Thereby small lumps evade detection and therefore delays diagnosis. That is why pregnant women with breast cancer are identified only after the tumor has extensively metastised.

To detect breast cancer in the early stage, self examination of breast by the pregnant and nursing women themselves is recommended. Women should be rendered clinical breast examinations during their routine antenatal visits. A mammogram may be considered as it poses minimal risk to the fetus. But, mammograms may sometimes not detect the lesions.

Choriocarcinoma is an extreme variant of trophoblastic disorders and is occasionally associated with a normal pregnancy. Intracerebral hemorrhage probably is the first manifestion of a n underdiagnosed gestational choriocarcinoma.

MULTIPLE SCLEROSIS (MS)

Multiple sclerosis is a neuroinflammatory disorder that involves defective myelination process. The disease may manifest with both motor and sensory deficits, loss of coordination, altered sensation visual symptoms that include double vision and nystagmus. The clinical variants of multiple sclerosis are relapsing-remitting, secondary progressive, primary progressive and progressive relapsing.

First and second trimesters seem to be more susceptible phase s for relapse. When there is an episode of relapse of multiple sclerosis in pregnancy, they present with tiredness, restless legs and urinary dysfunction. Should relapse occur, management is the same as for non-pregnant women.

Magnetic resonance imaging of the brain and spine demonstrate zones of demyelination (lesions or plaques).

If relapse is mild, it usually does not need any intervention, yet requires physiotherapy to overcome the increased disability levels in the intial phase. Intravenous administration or oral corticosteroids such as methylprednisolone for 3–5 days can hasten the recovery and brings about remission.

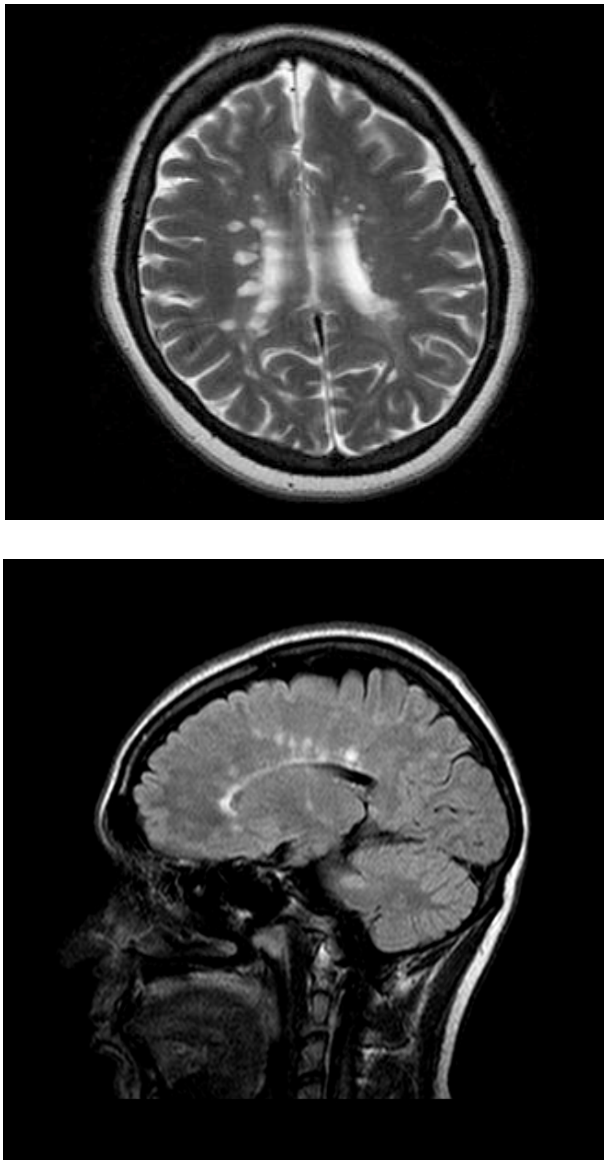


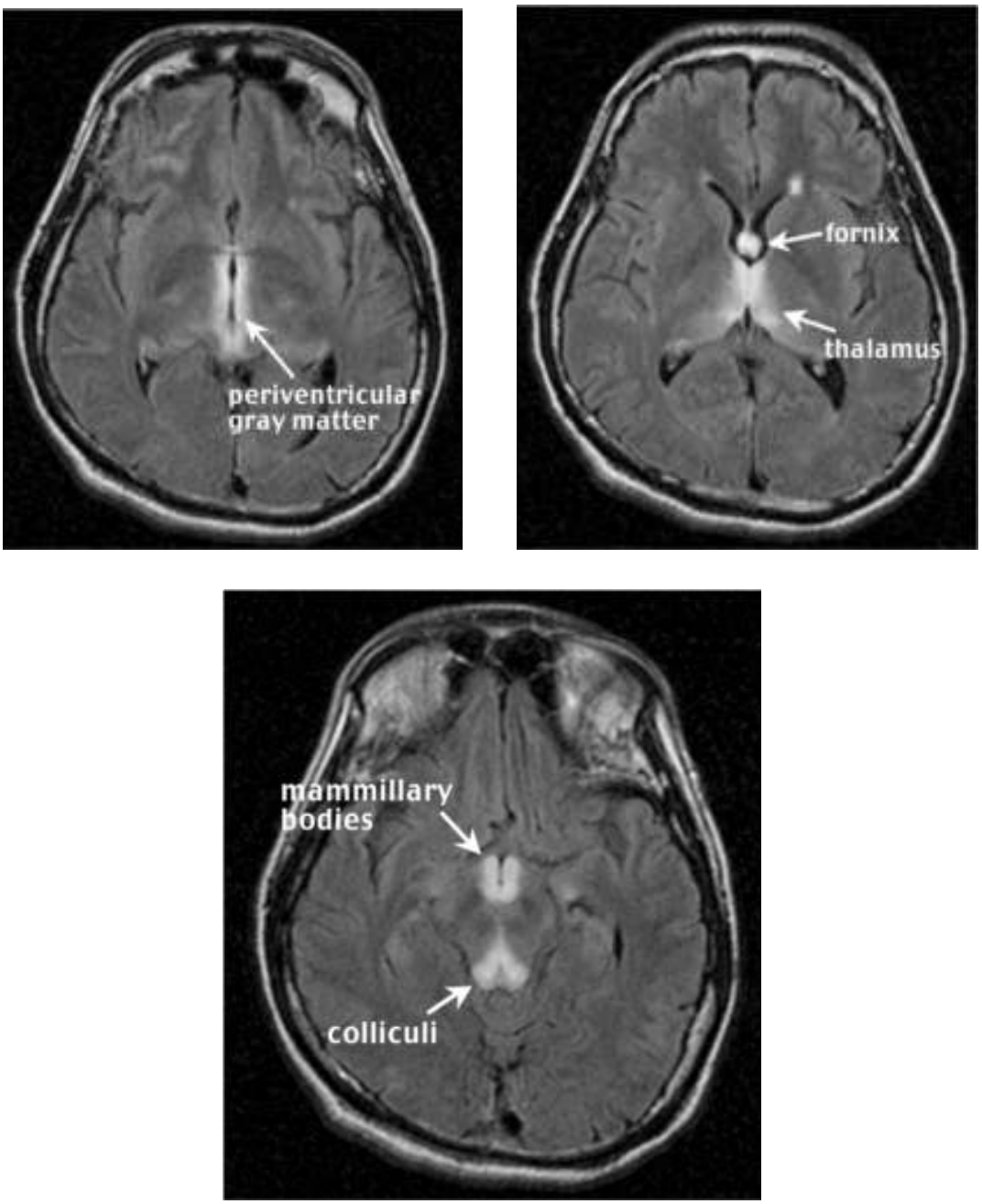
Figure – T2W axial and FLAIR sagittal image shows periventricular hyperintensity lesions with (dawson finger)appearance multiple sclerosis.

WERNICKE'S ENCEPHALOPATHY

Wernicke's encephalopathy is an extremely rare condition in pregnancy that follows intractable vomiting. There is an acute onset of confusion, double vision and ataxia. Neurological assessment demonstrates nystagmus and decreased sensation. MR imaging shows lesions in the mediodorsal nuclei of thalami on both sides with restricted diffusion in the posterior thalamus.

Likewise spontaneous hyperintensities in FLAIR and T 2 weighted images are visualized in the hypothalamus and also in the periaqueductal gray matter. These clinical features are in favour of diagnosis of Wernicke's encephalopathy. Intravenous administration of thiamine at a dosage of 300mg/day has shown a potentially favorable improvement.

Figure – Axial FlAIR images shows hyperintense signal in mammillary bodies, colliculi, periventricular gray matter, fornix and thamamus in a pregnant patient with intractable vomiting suggestive of Wernicke's encephalopathy.



CENTRAL PONTINE OSMOTIC DEMYELINOSIS

Pregnant women who are in the early trimester often present with hyperemesis gravidarum which are usually treated with intravenous fluids to combat dehydration and electrolyte imbalance. If hyponatremia is corrected within a short span of time, it is liable to triggerf an extremely rare yet a potentially perilous condition called central pontine osmotic demyelinosis. There is a sudden demyelination in the pons as well as in the extrapontine areas. Neurons in the cerebrum, cerebellum, basal ganglia and internal capsule also undergo demyelination

The myelinolysis of the corticospinal tract causes spastic variant of quadriparesis. When the corticobular tract is involved it results in pseudobulbar paralysis. Some of the typical symptoms include myalgia which becomes generalized and keeps progressing, cognitive dysfunction, seizures and rarely neuropsychiatric manifestations. These patients mostly recover well on treatment with Levodopa. Nevertheless, adequate care must be taken by health care personnel to avoid the occurrence of such medical emergencies.

HYPOTHESIS

This study focuses on imaging assessment of neurological symptoms in pregnancy and puerperium that are under diagnosed due to reluctance by the obstetricians to perform MRI and CT in pregnant women. Early diagnosis and prompt treatment can help bring down the mortality and morbidity in pregnancy and puerperium.

Using MRI 1.5 tesla, pregnant women have been evaluated, whereas both CT and MRI have been considered for puerperal patients. Using 4 slice spiral CT and 1.5 tesla MRI, classical imaging findings can be identified and thereby early diagnosis can be arrived at. However contrast enhanced CT or MR studies has not been performed in our study.

MATERIALS AND METHODS

STUDY DESIGN: Observational study.

PATIENT SELECTION:

Consecutive pregnant and puerperal patients with neurological symptoms admitted to obstetrics and neurology ward, from the period of august 2013 to august 2014.

INCLUSION CRITERIA:

Patients who present with various neurological and recent onset neuro-psychiatric symptoms in pregnancy and puerperium;

Example:

- Seizures
- Altered sensorium
- Sensory-motor deficit
- Lactation failure
- Headache with other neurological symptoms

EXCLUSION CRITERIA:

- Known seizure disorder patients
- Known psychiatric patients
- Post traumatic patients

TECHNIQUES:

Antenatal period: MR imaging only

Post natal period: CT and MR imaging

- Relevant history of the patient will be obtained based on expert's opinion

CT imaging during post natal period:

- Non-contrast CT imaging of the head with 4 slice CT scanner machine (Toshiba - Alexion)
- Post intravenous contrast enhanced CT study of head (if indicated ie in post – natal patients)

  Contrast dose – 2 ml / kg, of Iohexol, 360 mg/ml

  Image acquired in parenchymal phase as routine.

  Parameters

Slice thickness = 3mm.

Pitch factor = 1.2mm.

Reconstruction interval

- Infratentorial compartment 3 mm to 5 mm.
- Supratentorial compartment 5 mm to 7 mm.

MR imaging during both antenatal and postnatal periods

- Magnetic resonance imaging of brain with 1.5 tesla (Siemens, Symphony).

  Routine Protocol:

  T1 weighted imaging - Sagittal,

  T2 weighted imaging - Axial,

  Fluid attenuated inversion - Coronal,

  Gradient echo images - Axial,

  Diffusion weighted images and apparent diffusion coefficient - Axial.

  MR arteriogram and venogram.

OBSERVATIONS AND RESULTS

- Total number of cases of pregnancy and puerperium related neurological diseases (CVT, PRES, PCA and Arterial Infarctions) that occurred in our study period is 50. Total number of deliveries in our hospital in this study period is 7100.
- Incidence rate of neurological diseases is 704/100000 deliveries.
- Total number of Cerebral venous thrombosis cases that occurred in our study period is 35.
- Incidence rate of CVT cases is 492/100000 deliveries.
- Total number of posterior reversible encephalopathy cases that occurred in our study period is 12.
- Incidence rate of PRES cases is 169/1000000 deliveries.
- Only 2 cases of arterial infarctions occurred in our study period.
- Incidence rate of arterial infarctions is 28/100000 deliveries.
- Total number of deaths that occurred in our study period among the 50 cases is 4.

- Mortality rate is 56/100000 deliveries.
- All four cases of mortality occurred due to venous occlusion followed by hemorrhagic infarctions.
- Totally 120 patients with neurological symptoms were subjected to imaging;

  i) In 78 post natal cases both CT and MRI were done.

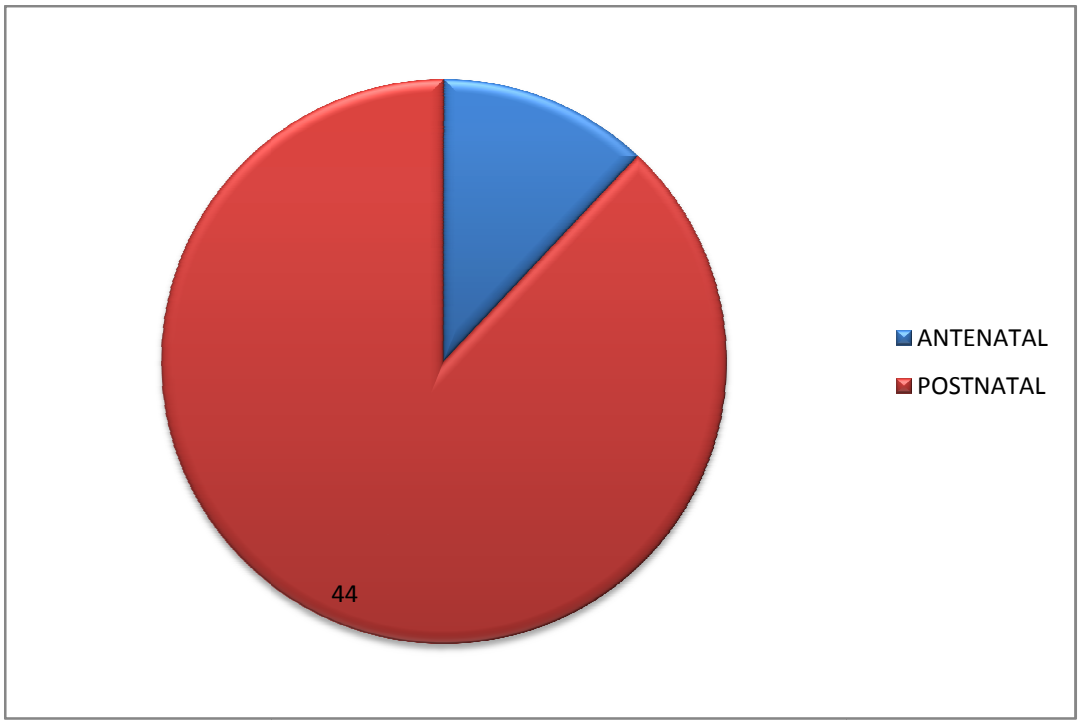
  ii) In 18 post natal cases CT scan was done.

  iii) In 24 antenatal cases MRI was done.

- Most common symptom associated with positive imaging findings is acute headache.
- Symptoms which predict poor prognosis are altered sensorium and motor weakness.
- Most commonly involved venous sinuses in CVT are superior sagittal sinus and sigmoid sinus.

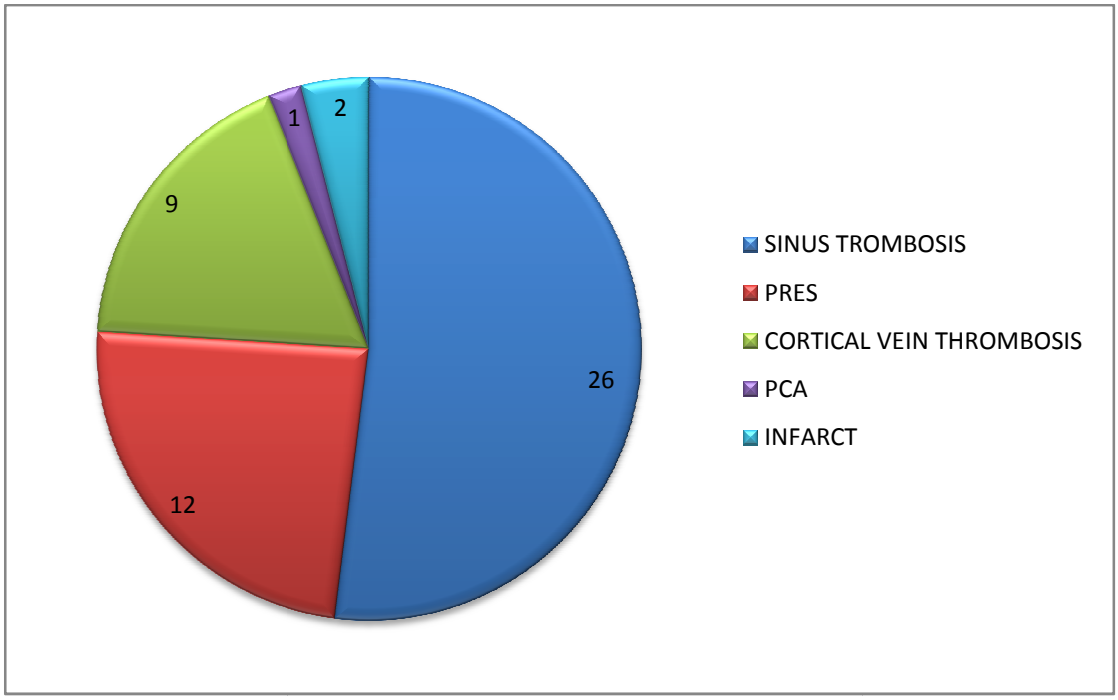
NEUROLOGICAL DISEASES IN PREGNANCY PERIOD VS PUERPERIUM

Diagnosis	TOTAL
ANTENATAL	6
POSTNATAL	44
TOTAL	50



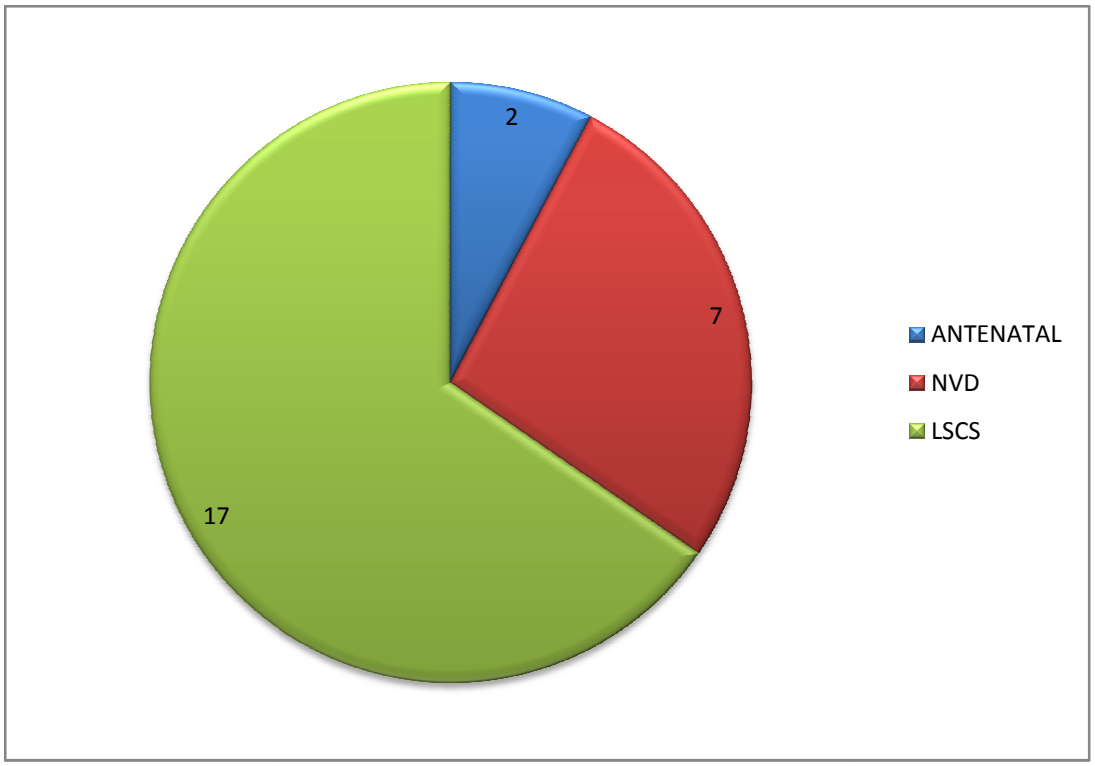
SPECTRUM OF NEUROLOGICAL DISEASES IN CMCH

Diagnosis	TOTAL
VENOUS SINUS THROMBOSIS	26
PRES	12
CORTICAL VEIN THROMBOSIS	9
PCA	1
INFARCT	2
TOTAL	50



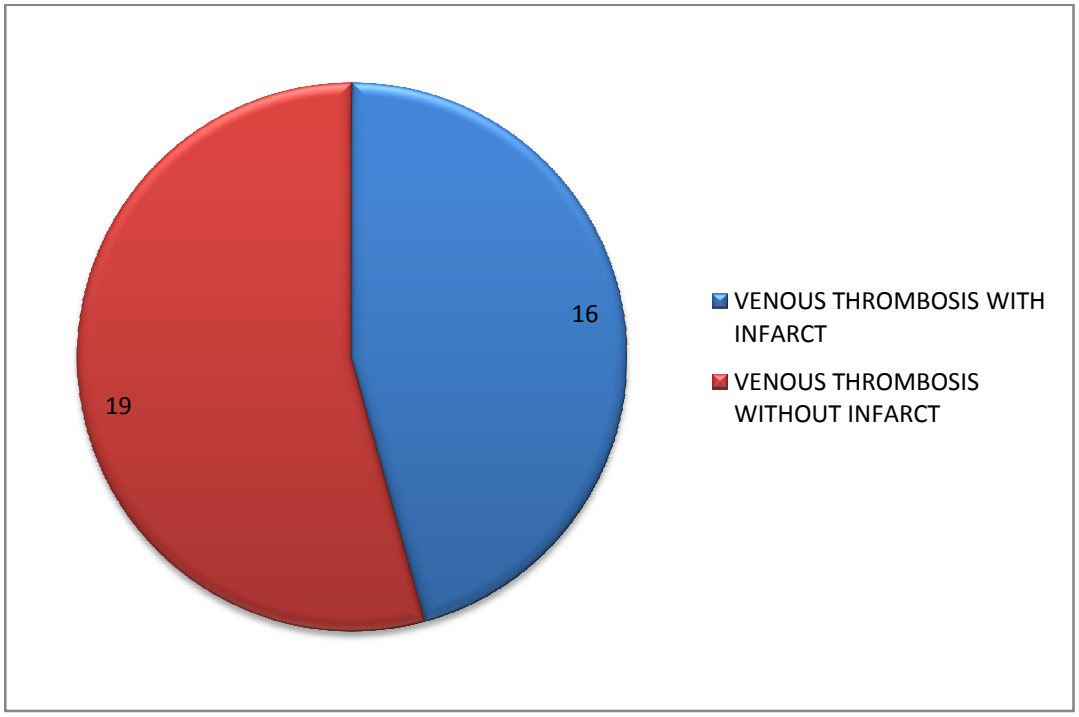
CEREBRAL VENOUS SINUS THROMBOSIS IN PREGNANCY AND PUERPERIUM

Diagnosis	TOTAL
ANTENATAL	2
NVD	7
LSCS	17
TOTAL	26



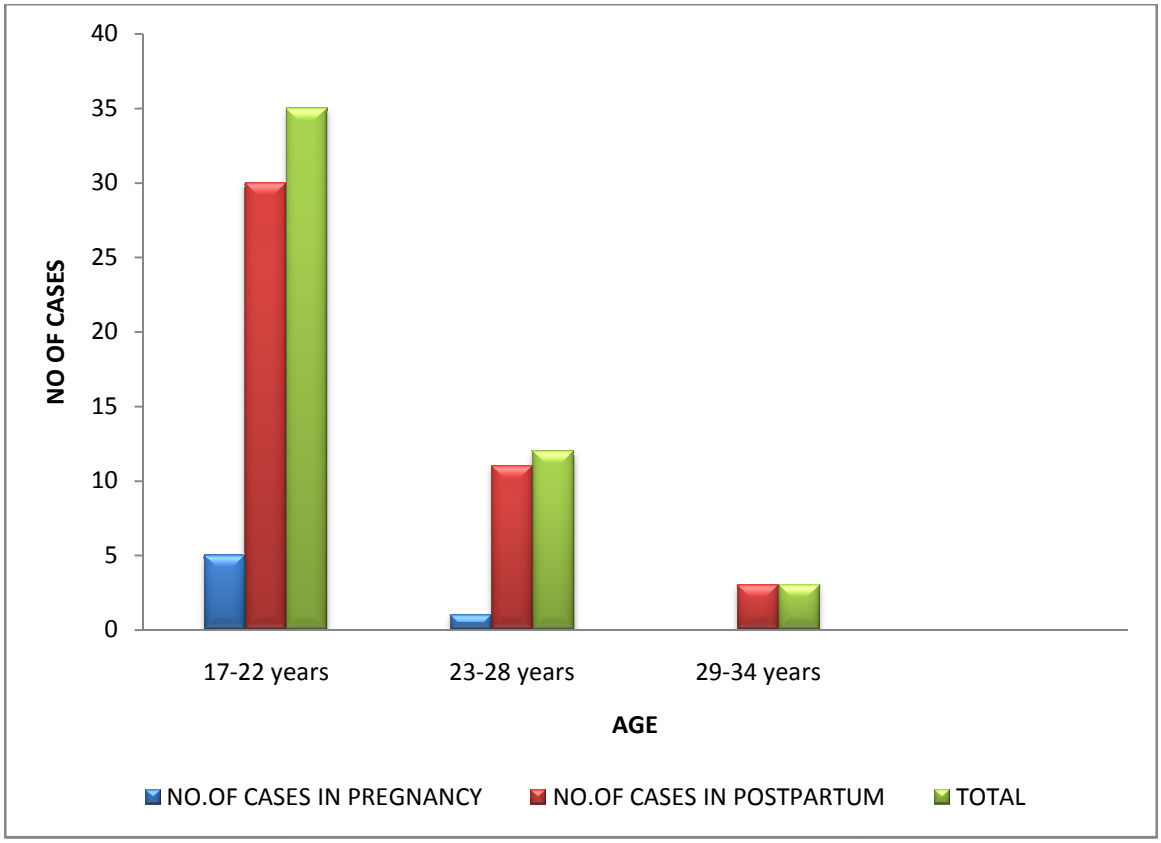
INCIDENCE OF HEMORRHAGIC INFARCT IN CVT

Diagnosis	TOTAL
VENOUS THROMBOSIS WITH HEMORRHAGIC INFARCT	16
VENOUS THROMBOSIS WITHOUT HEMORRHAGIC INFARCT	19
TOTAL	35



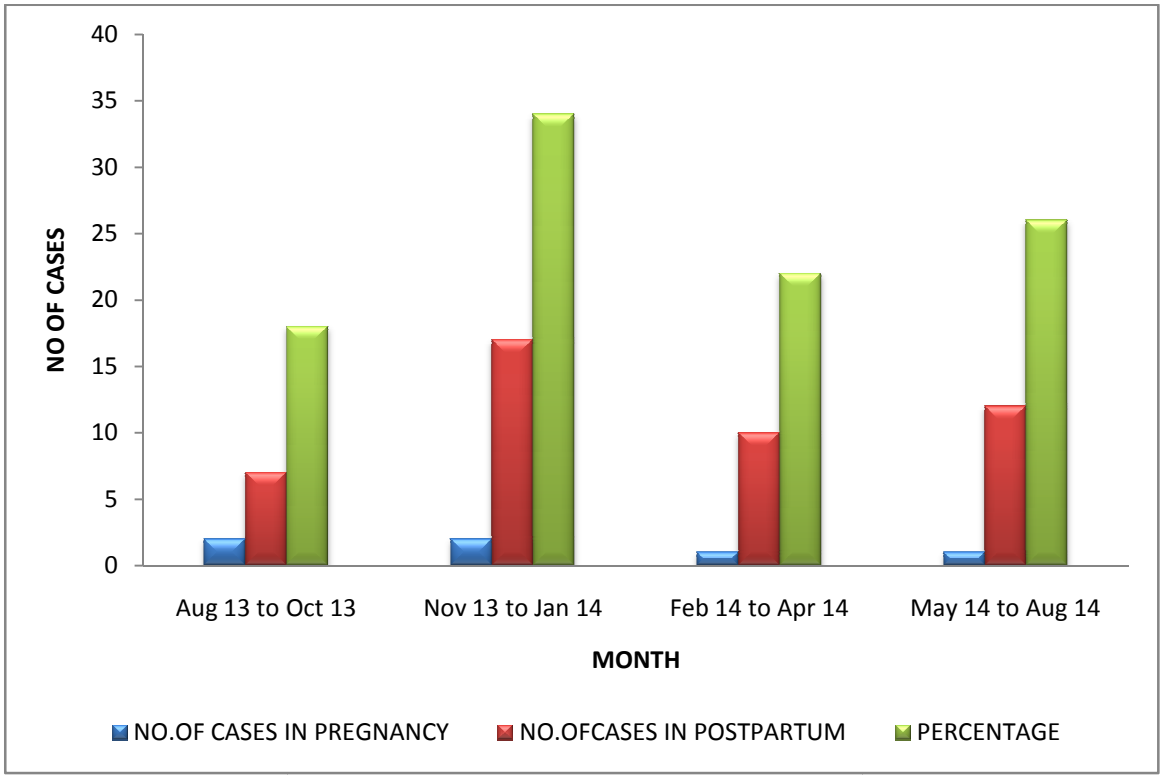
AGE DISTRIBUTION OF CASES

Age group	*No. of cases in pregnancy*	*No. of cases in postpartum*	*Total*
17 to 22 years	5	30	35
23 to 28 years	1	11	12
29 to 34 years	-	3	3



PERIOD DISTRIBUTION OF CASES

Period of incidence	*No. of cases in pregnancy*	*No. of cases in postpartum*	*Percentage*
Aug 2013 to Oct 2013	2	7	18
Nov 2013 to Jan 2014	2	17	34
Feb 2014 to Apr 2014	1	10	22
May 2014 to Aug 2014	1	12	26



PERIOD OF DISTRIBUTION AND MORTALITY

Period of incidence	No. of cases in pregnancy	Mortality	No. of cases in postpartum	Mortality
Aug 2013 to Oct 2013	2	-	7	-
Nov 2013 to Jan 2014	2	-	17	2
Feb 2014 to Apr 2014	1	-	10	1

SYMPTOM ANALYSIS

Symptoms	*No. of cases in pregnancy*	*No. of cases in postpartum*	*Total*
Acute Severe Headache	4	19	23
Seizures	3	9	12
Focal neurological deficit	-	17	17
Altered sensorium	-	5	5

INCIDENCE IN NORMAL Vs LSCS DELIVERY

INCIDENCE OF NEUROLOGICAL DISEASE		
No. of cases in pregnancy	***No. of cases in postpartum***	
	Normal delivery	Caesarean delivery
6	13	31

CT Vs MRI IN DETECTION OF SUPERFICIAL CORTICAL VEIN THROMBOSIS

DIAGNOSIS OF SUPERFICIAL CORTICAL VEIN THROMBOSIS		
	Detectd by CT	Detected by MRI
Pregnancy	-	-
Postpartum	-	9

PERCENTAGE OF CASES

Neurologic Disease	No of cases	Percentage
Cerebral venous thrombosis	35	70%
Posterior Reversible encephalopathy Syndrome (PRES)	12	24%
Hemorrhagic infarct	15	30%

VENOUS SINUS Vs SUPERFICIAL CORTICAL VEIN INVOLVEMENT

	No of cases	***Percentage***
Venous Sinus	*26*	*52%*
Superficial Cortical Vein	*9*	*18%*
Total	***35***	

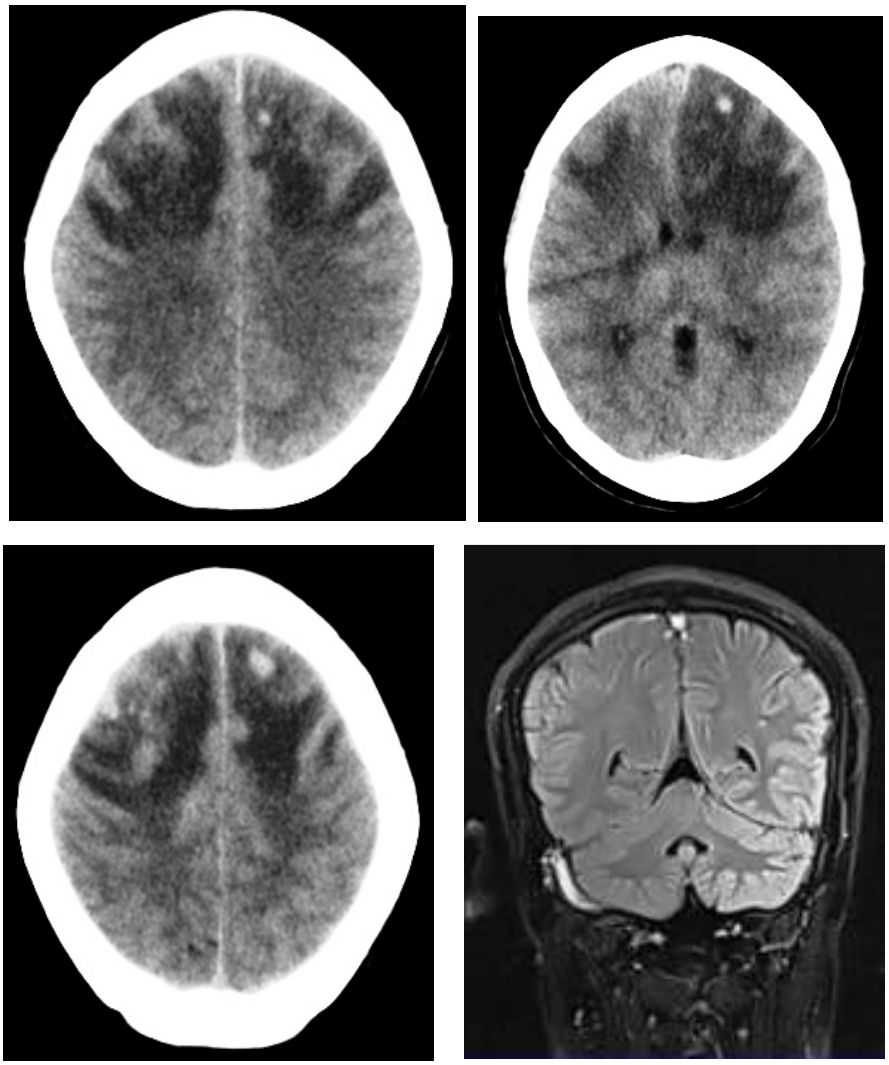
Among sinus thrombosis cases frequency of involvement of individual venous sinuses

- Superior sagittal sinus – 16 cases.
- Sigmoid sinus – 16 cases.
- Transverse sinus – 9 cases.
- Straight sinus – 4 cases.

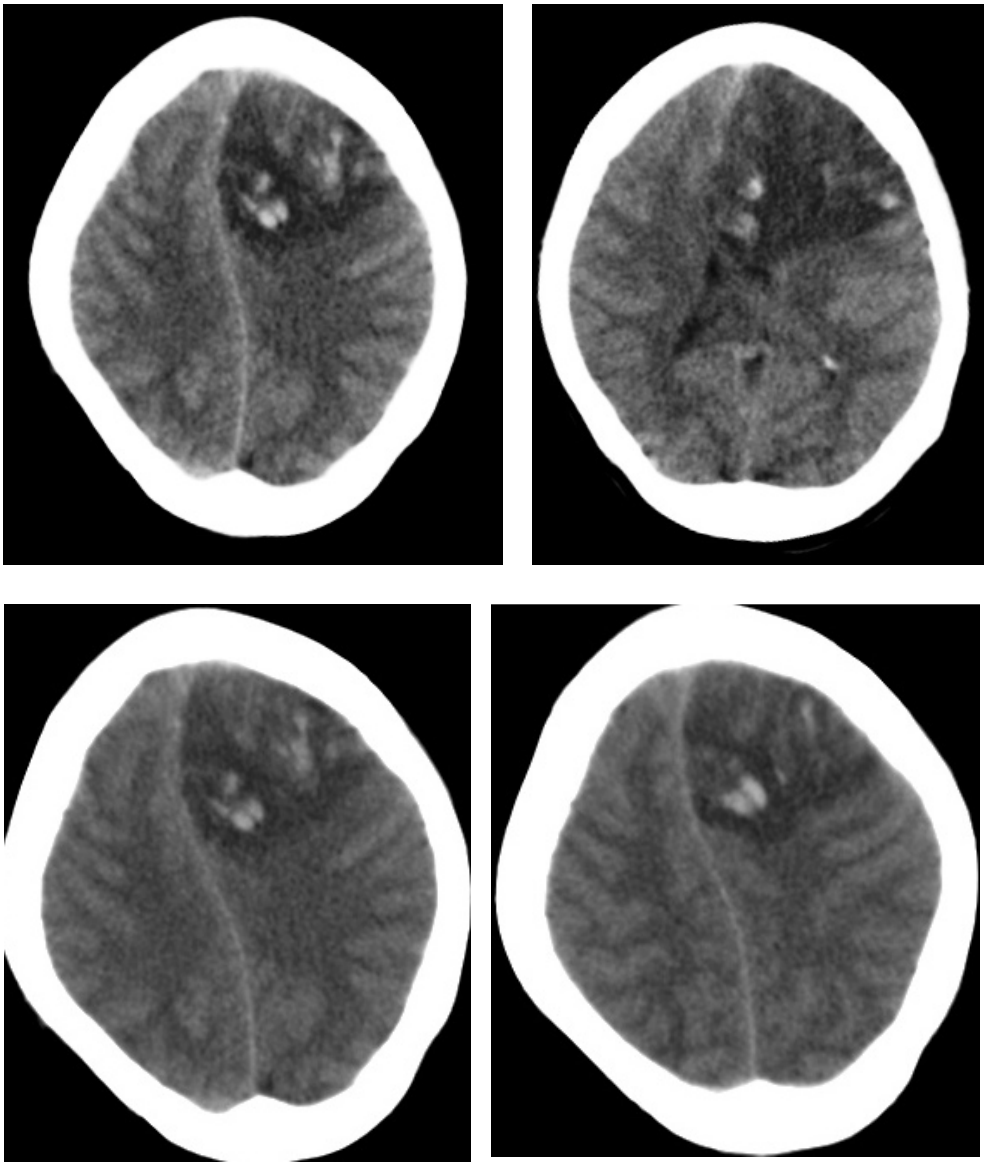
REPRESENTATIVE CASES

There were four cases of mortality during the study period from Aug 2013 to Aug 2014.

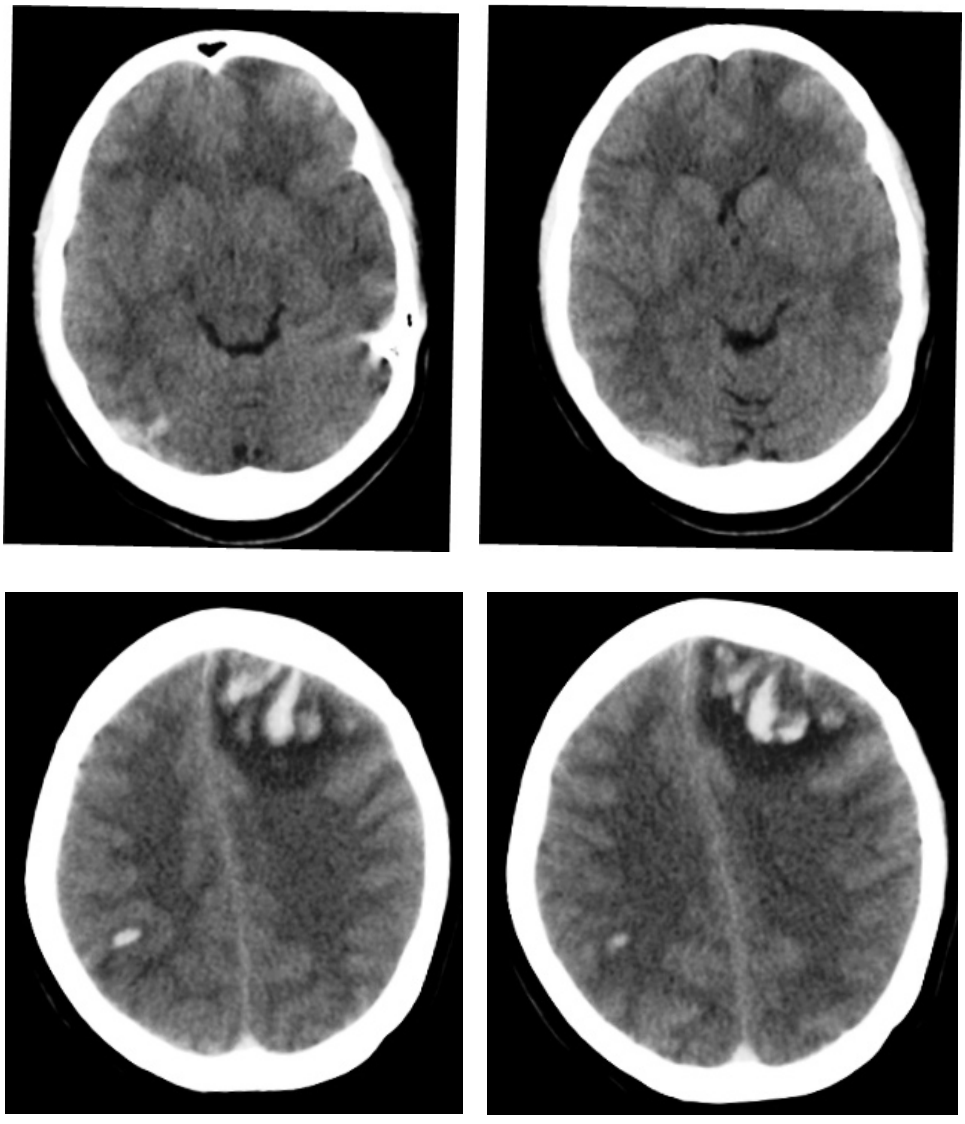
Case I – 20 years old primi, post LSCS developed seizures and motor weakness. Imaging with CT and MRI showed sigmoid transverse superior sinus thrombosis with bilateral frontoparietal hemorrhagic infarcts.



Case II – 29 years old, post LSCS in immediate post natal period presented with altered sensorium. CT showed bilateral frontal hemorrhagic infarcts and midline shift to right side. MRI showed superior sagittal sinus thrombosis.

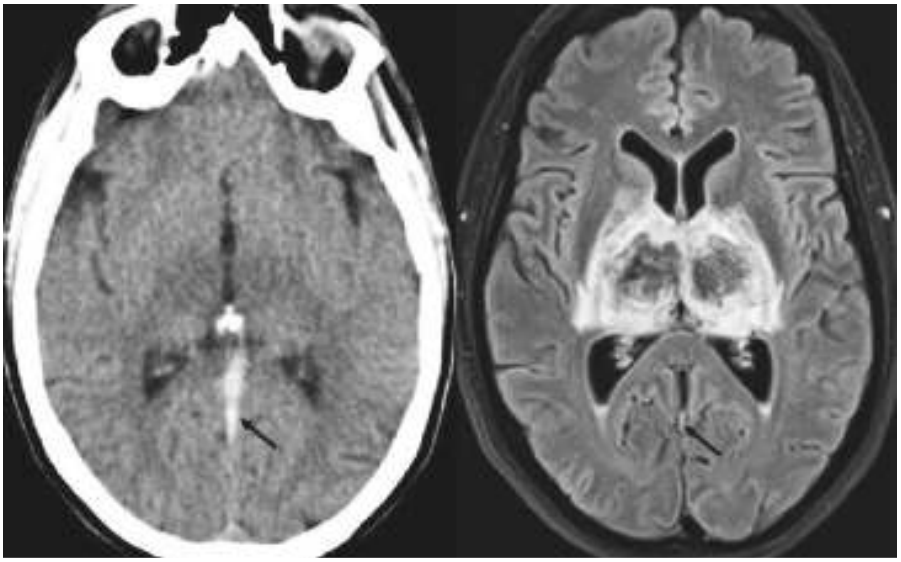


Case III - 27 years old, post LSCS – 24 hours after delivery, presented with altered sensorium and motor weakness. CT showed right parietal and left frontal hemorrhagic infarcts. MRI showed thrombus in the right sigmoid, transverse, superior sagittal sinuses and also in the venous confluence.

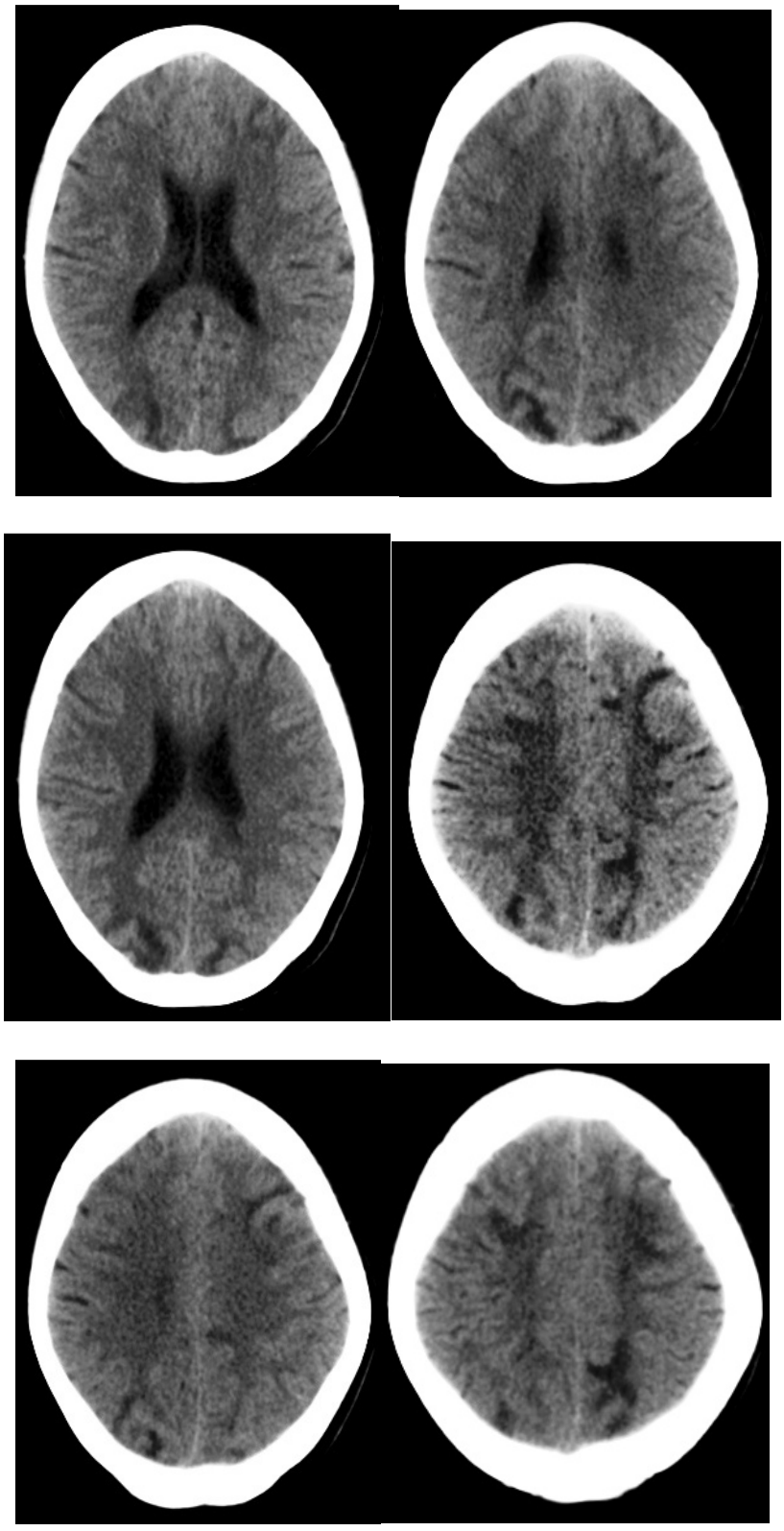


Case IV – 19 years old primi after normal vaginal delivery presented with status epilepticus. CT and MRI showed straight sinus and superior sagittal sinus thrombosis with bilateral thalamic hemorrhagic infarcts.

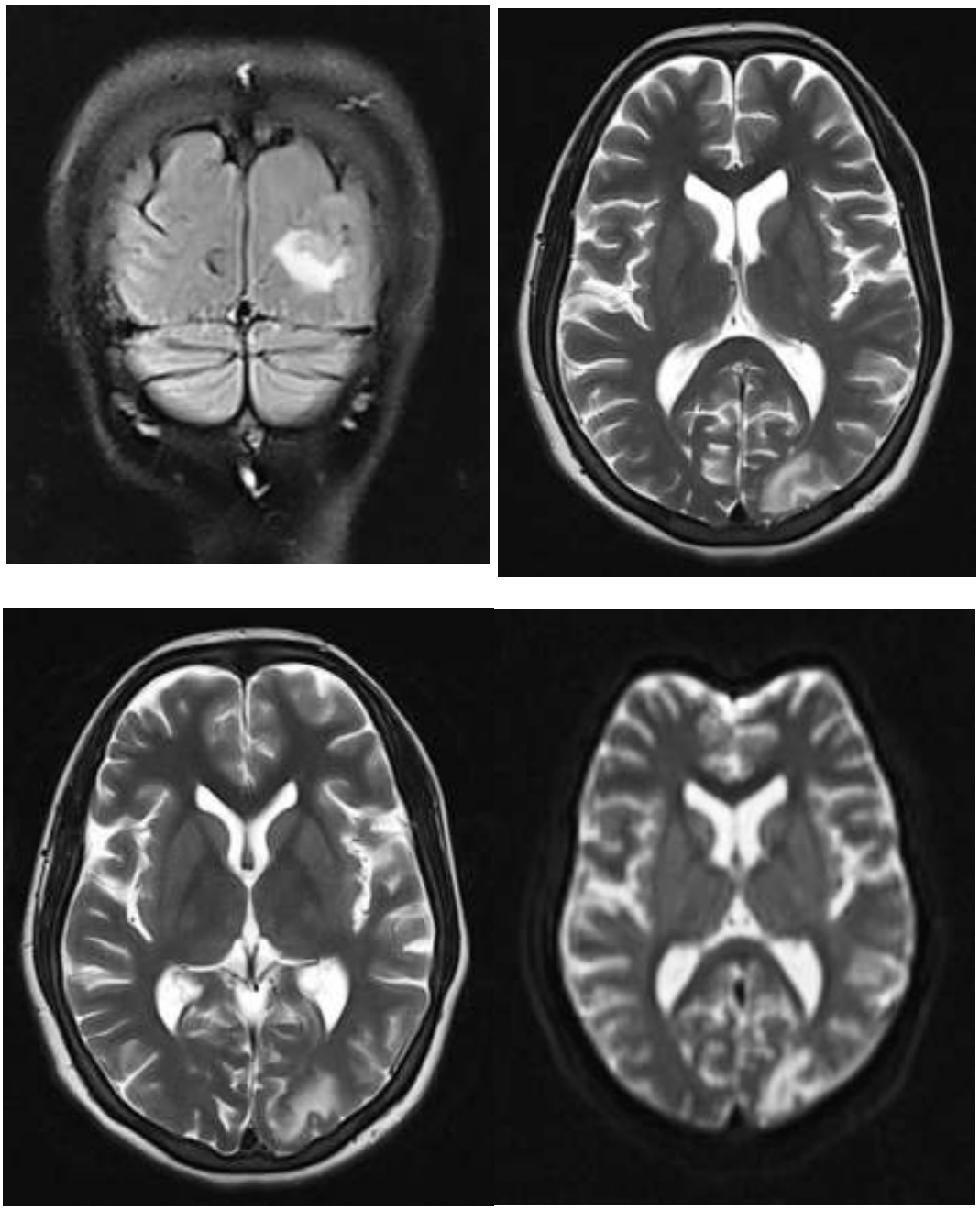
CT and MR Shows deep vein thrombosis and bilateral thalamic hemorrhagic infarct.



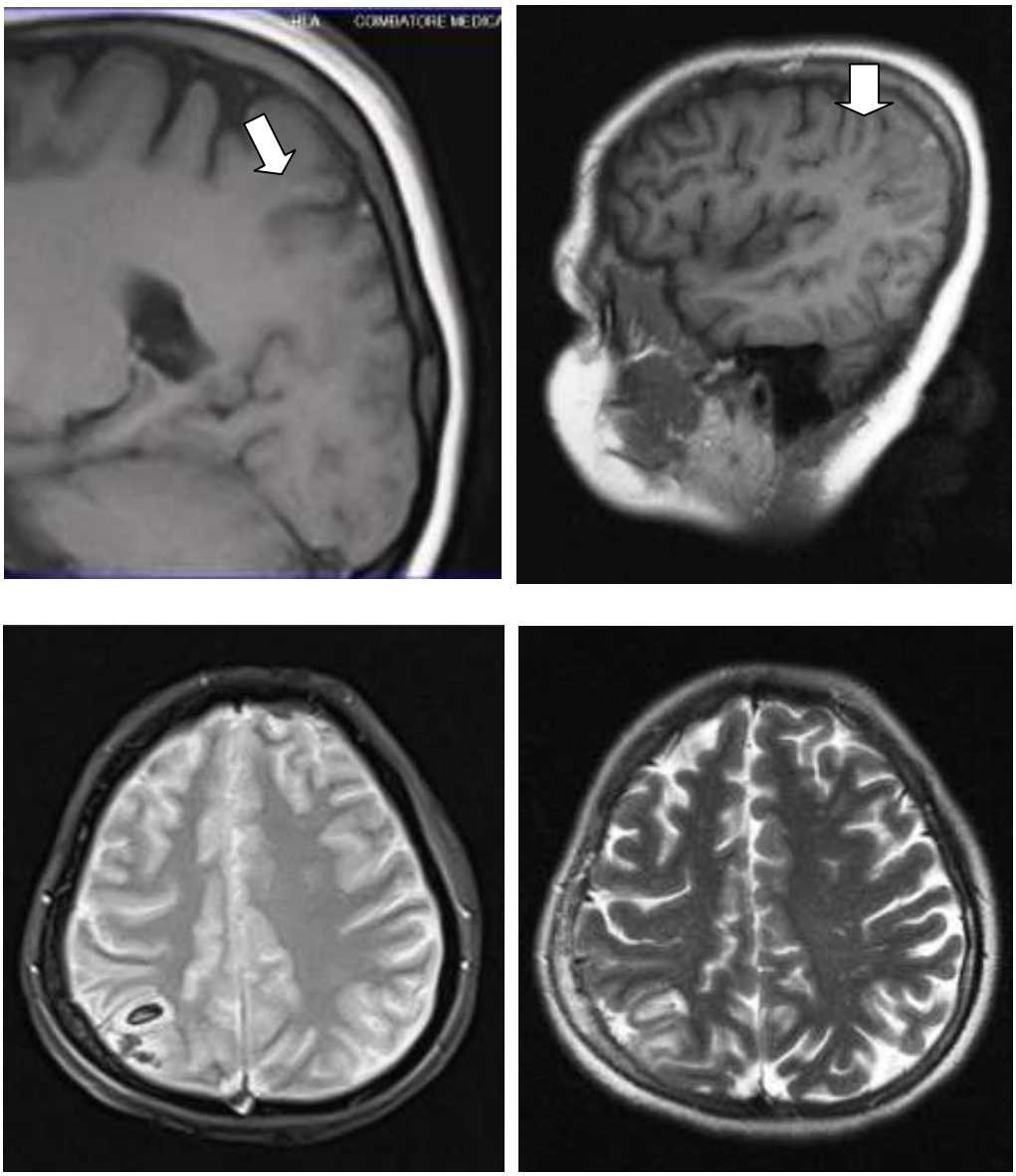
Case V – 26 year old, in the immediate postpartum period presented with features of PRES



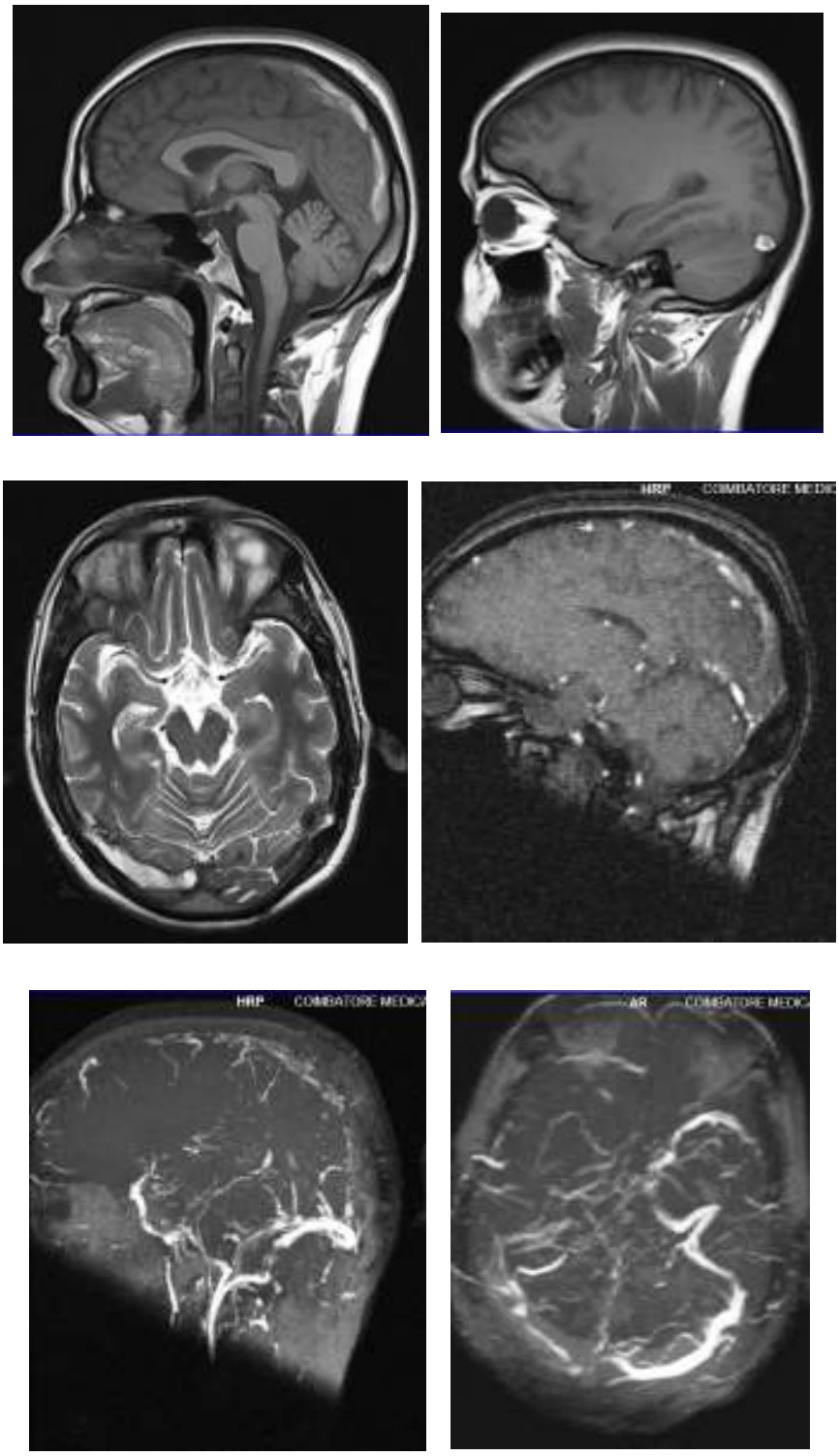
Case VI – Antenatal 8 months of amenorrhoea presented with headache. T2W and flair images show parietooccipital white matter hyperintensity. There is no diffusion restriction – which is suggestive of PRES



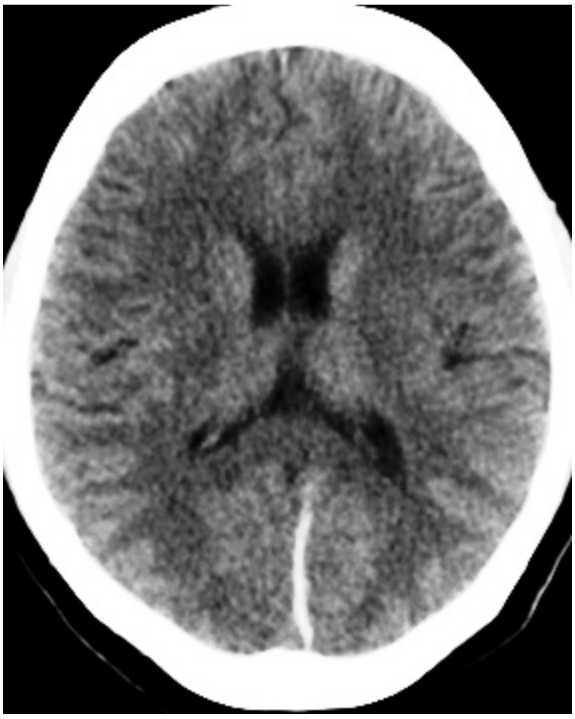
Case VII – 19 years AN patient, T1W images sagittal sections show superficial cortical vein thrombus T2W and GRE images show hemorrhagic infarcts.



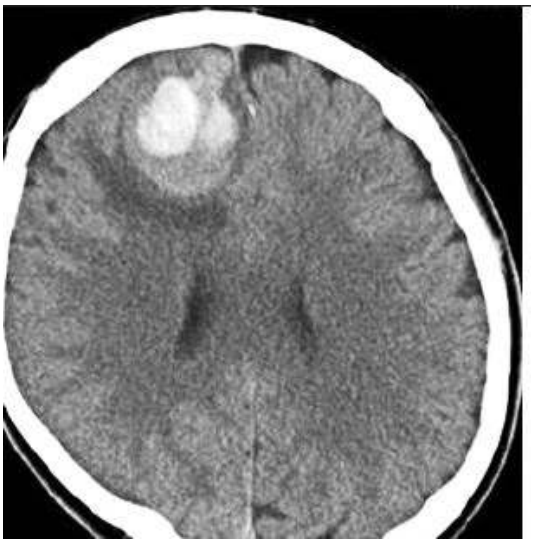
Case VIII – 23 yrs postpartum patient, presented with severe headache and seizures. T1 sagittal T2 axial and MRV show SSS, right transverse and sigmoid sinus thrombosis.

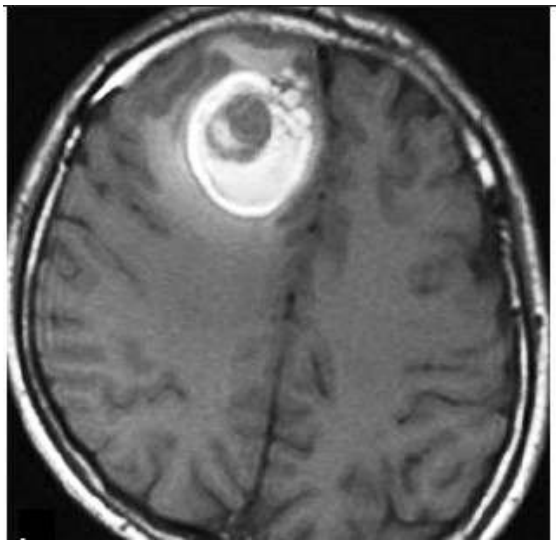


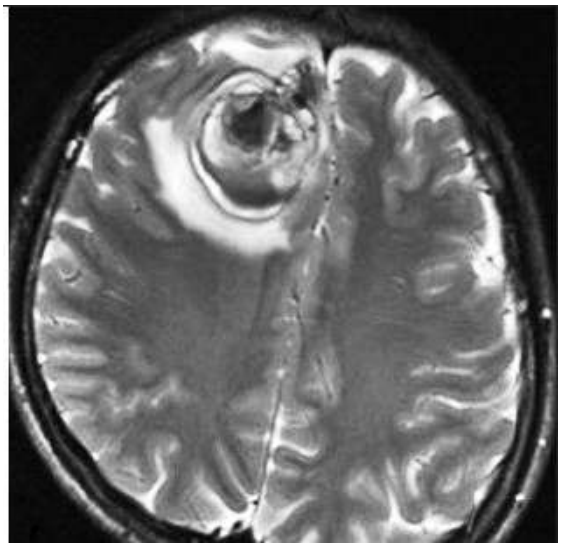
Case IX- 22 yrs old patient presented with severe headache and seizures in the postpartum period. Ct scan show straight sinus thrombosis.



CASE X – 22 yrs old, postpartum normotensive women with motor weakness and headache. Imaging show right frontal intracerebral hematoma without any evidence of venous thrombosis - suggestive of PCA.







DISCUSSION

This study focuses on neurological illnesses that develop during pregnancy and puerperium and excludes pre-existing neurological and psychiatric illnesses.

Among the clinical presentations and symptoms, cases presenting with severe headache along with other neurological signs often end up with positive neuroimaging findings.

There were fifty pregnancy and puerperium related neurological disease encountered in Coimbatore Medical College Hospital from august 2013 to august 2014

Commonest pregnancy related disease found to occur in CMCH was cerebral venous sinus thrombosis. The incidence rate of CVT cases 492/100000 deliveries.

Second common neurological disease is posterior reversible encephalopathy syndrome. Incidence rate of PRES cases 169/1000000 deliveries.

Most of these neurological diseases are found to occur in the postpartum period. In general, it can be concluded that mortality due to

neurological complications of pregnancy and puerperium is very unusual. During this study period four cases of mortality occurred.

Four cases of mortality were due to large hemorrhagic infarcts with midline shift that were identified to be an aftermath of cerebral vein thrombosis. Surgical intervention in one of the patients with massive midline shift, by craniotomy and decompression of hematoma was attempted. But the patient did not survive.

All the PRES cases which were seen in this study period eventually recovered completely. Most common area of involvement in PRES is the parieto-occipital area of the brain. There is no mortality related to any of the PRES cases.

The diagnosis of cortical superficial vein thrombosis has been always made by Magnetic Resonance Imaging only.

CT mostly does not help to diagnose cortical superficial vein thrombosis as such but enables to detect hemorrhagic infarcts in the parenchyma which is the consequence of the cortical vein thrombosis. However, T1 weight images of MRI brain is superior to CT in demonstrating cortical vein thrombus is almost in all cases.

In western countries most pregnancy related infarctions are reportedly arterial in origin. Venous infarctions are not common in western countries. But in India studies show decreased incidence of arterial occlusions in pregnancy related infarctions. In our study by neuro imaging only 2 cases of arterial occlusions have been detected.

Thus venous infarctions are more common in India. Probably this could be correlated with the traditional practices that are followed in India as a routine for post natal care such as withholding of fluids and climatic conditions. Also it could be attributed to increased prevalence of anemia and sepsis in India.

In our study, patients who presented with venous thrombosis followed by hemorrhagic infarctions show poor prognosis. Western studies and other Indian studies also shows poor prognosis in cases with hemorrhagic infarctions of pregnancy.

In our study maternal mortality rate with neurological disease is 4 cases. (56/100000 deliveries 0.056 %).

Incidence of pregnancy related cerebrovascular diseases are different in various parts of the world. Westerns study shows of 34.2/100000 deliveries(USA based study jane AH etal 2005).

Chennai based study in 2012 shows incidence of 66/100000 deliveries (J obstet. Gynec India Apr 2013. T.Radha bai Prabhu).

Risk of neurological diseases vary with the stages of pregnancy and postpartum. In our study clustering of PRES cases occurred in late pregnancy period and early postpartum period.

Most often PRES cases demonstrate a dramatic resolution of lesion. The stage of recovery can be speculated with the help of MRI with diffusion weighted images, by the absence of restricted diffusion.

It has been understood that the presenting symptoms of neurological disease are also relevant in determining the outcome. Those cases presenting with the symptoms of altered sensorium and motor weakness are associated with poor prognosis.

Post natal cases are more inclined to develop cerebral venous thrombosis than antenatal cases. Further, among the post natal cases women who have undergone cesarean section are more liable to present with cerebral venous thrombosis than those women who have delivered normally.

Incidence of neurological diseases are seemingly high in our study population, when compared to national and studies based on the population in the developed countries. This may be due to the fact that Coimbatore Medical College Hospital is the tertiary care centre for the district of Coimbatore which deals with referred cases from rural and urban primary health centres.

Without taking into consideration the referrals that account for half of the total number of cases, incidence rates are apparently halved as regards the neurological disease. Despite this deduction of the number of referral patients from the total number of cases, the occurrence rates were still high when compared to the national average.

Common occurrence of neurological diseases in our study falls in the age group of 17 to 22 years.

A significant aspect of neuroimaging is that accurate diagnosis helps to advocate appropriate treatment strategies. For instance, cases with ischemic stroke which can be confirmed by imaging are benefitted by early initiation of thrombolytic measures. Also treatment of hemorrhagic venous infarction involves use of anticoagulants whereas it is contraindicated in treatment of primary intracerebral hemorrhage (due to transient intrapartum hypertension).

CONCLUSION

Neurological symptoms are not uncommon during pregnancy and puerperium. While most symptoms turn out to be benign, in some patients they may indicate serious underlying problem. Use of prompt and appropriate imaging modality potentially helps to diagnose serious illnesses earlier and more accurately, thus helping the obstetrician to institute appropriate treatment strategies. This has a definite impact in reducing maternal morbidity and mortality.

SUMMARY

Imaging abnormalities in the CNS are produced by a constellation of disorders that seem to coexist with pregnancy and puerperium. Some are attributed to the physiologic aspects of reproduction, commonest being eclampsia, whereas others are part of pathophysiologic conditions noted in the general population that is unusually aggravated in pregnant women, like dural sinus thrombosis.

Based on the observation study conducted at Coimbatore Medical College Hospital, cerebral venous thrombosis is the commonest neurological entity that has been diagnosed by imaging. 35 cases of CVT were diagnosed among the 50 patients. Apart from this, 12 cases were identified as posterior reversible encephalopathy syndrome. Only two cases of arterial occlusion related neurological diseases were seen in the stipulated study period.

Unfortunately, there were four cases of mortality in our study which were due to venous occlusion followed by hemorrhagic infarcts.

Identification of the unique imaging findings in eclampsia enables to rule out life threatening conditions such as stroke.

PCA vasculitis is another entity that should be considered in addition to the other common causes of intracerebral hemorrhage. In addition, early diagnosis is the prime motive so as to prevent untoward consequences of delayed diagnosis. CT and MR venography enable early noninterventional diagnosis of CVT.

CONSENT FORM

Yourself Mrs …….. are being asked to be a participant in the research study titled **CT and MR Imaging of Neurological diseases in Pregnancy and Puerperium** in CMC hospital, Coimbatore, Conducted by **Dr.T.PRINCE JEBA ANAND**, Post Graduate student in the department of radiology, Coimbatore Medical College Hospital. You satisfy eligibility as per the inclusion criteria. You can ask any questions you may have before agreeing to participate.

PURPOSE OF RESEARCH

- CT and MR imaging characterization of the various cerebral parenchymal, cerebrovascular, neuro–endocrine and neoplastic disorders affecting the central nervous system during pregnancy and the puerperium of patients admitted to obstetrics and neurology ward of Coimbatore medical college hospital, Coimbatore.
- To know the prevalence of disorders affecting the central nervous system during pregnancy and the puerperium.

PROCEDURES INVOLVED

- Detailed history regarding risk factors symptoms and signs of neurological diseases of pregnancy and puerperium will be recorded.

- Non-contrast CT imaging of the head– for puerperal patients (with precaution for antenatal patients-Lead apron etc.)

- Post intravenous contrast enhanced CT study of head if indicated – for puerperal patients.

- Magnetic resonance imaging of brain with 1.5 tesla (Siemens, Symphony) – for antenatal and puerperal patients. Routine Protocol

- MR arteriogram and venogram.

- MR proton spectroscopy (Single and Multivoxel) – if necessary.

PRIVACY AND CONFIDENTIALITY

Privacy of individuals will be respected and any information provided will be kept confidential.

AUTHORIZATION TO PUBLISH RESULTS

Results of the study may be published for scientific purposes and / or presented to scientific groups; however you will not be identified.

STATEMENT OF CONSENT

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me and I may ask questions at any time.

…….

Signature / left thumb impression of volunteer with date

……..

Signature of witness with date

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MASTER CHART

S.No	Age	NVD/LSCS	AN/PN	Symptoms	CT findings	Mri findings	Diagnosis	Prognosis
1	19	AN	AN 8 MONTHS	Headache,Blurred Vision	not done	T2,FLAIR-hyperintensity in lt parietooccipital region-PRES	PRES	good
2	25	LSCS	PN 3 DAYS	Weakness Lt Ul & Ll	Haemorrhagic infarct rt frontal lobe	Cortical vein thrombosis	cortical vein thrombosis	good
3	26	LSCS	IMMEDIATE PN	Headache,Blurred Vision	B/L frontoparietooccipital white matter edema-PRES	not done	PRES	good
4	29	LSCS	IMMEDIATE PN	Altered Sensorium	B/L frontal haemorrhagic infarct,midline shift to rt	SSS Thrombosis with B/L haemorrhagic frontal infarct	sinus Thrombosis	bad
5	20	LSCS	IMMEDIATE PN	Seizures,Weakness	hyperdense rt sigmoid,transverse,confluence & sss	hyperintensity in the sinuses with b/l frontoparietal hagic infarct	sinus Thrombosis	bad
6	22	NVD	PN 1 DAY	Headache,Weakness Rt Ul & Ll	Lt frontal haemorrhagic infarct,midline shift to rt	Lt frontal haemorrhagic infarct with SSS thrombosis	sinus thrombosis	good
7	22	LSCS	PN 2 DAYS	Headache	hyperdense rt sigmoid,transverse & sup sagital sinus	hyperintense rt sigmoid,transverse,sss	sinus thrombosis	good
8	20	LSCS	PN 5 DAYS	Headache	hyperdensity in lt sigmoid sinus	hyperintensity IN Lt sigmoid sinus	sinus thrombosis	good
9	25	NVD	PN 3 DAYS	Weakess Rt Ul & Ll	lt parietooccipital infarct	not done	Arterial infarct	good
10	17	LSCS	PN 2 DAYS	Weakness Lt Ul & Ll	rt frontal hagic infarct with sss hyperensity	rt frontal hagic infarct with hyperintense SSS	sinus thrombosis	good
11	18	LSCS	1 DAY	Weakness Lt Ul & Ll	rt frontal hagic infarct	T1 hyperintense cortical vein with rt frontal hagic infarct	cortical vein thrombosis	good
12	20	AN	AN 8 MONTHS	Headache,Seizures	not done	b/l parietooccipital white matter hyperintensity on T2/FLAIR	PRES	good
13	21	LSCS	PN 2 DAYS	Headache	b/l parietooccipital edema-PRES	not done	PRES	good
14	24	NVD	PN 7 DAYS	Headache	hyperdensity in sss	not done	sinus thrombosis	good
15	19	LSCS	IMMEDIATE PN	Seizures	b/l parietooccipito white matter edema-PRES	not done	PRES	good
16	18	AN	AN 8 MONTHS	Seizures	not done	B/L frontoparietooccipital white matter edema-PRES	PRES	good
17	22	LSCS	PN 5 DAYS	Headache	hyperdense straight and sigmoid sinus	not done	sinus Thrombosis	good
18	23	LSCS	IMMEDIATE PN	Blurred Vision,Headache	B/L frontoparietooccipital white matter edema-PRES	B/L parietooccipital white matter edema-PRES	PRES	good
19	27	LSCS	PN 1 DAY	Weakness,Altered Sensorium	rt parietal & lt frontal hagic infarct	rt sigmoid,transverse,sss & confluence appear hyperintense	sinus Thrombosis	bad

S.No	Age	NVD/LSCS	AN/PN	Symptoms	CT findings	Mri findings	Diagnosis	Prognosis
20	26	LSCS	PN 1 DAY	Headache	hyperdense lt straight,transverse and sss	not done	sinus Thrombosis	good
21	19	LSCS	PN 1 DAY	Weakness Lt Ul & Ll,Headcahe	hagic infarct in rt frontal lobe with midline shift to lt	hyperintense rt sigmoid & transverse sinus with hagic infarct as on ct	sinus Thrombosis	good
22	21	LSCS	PN 3 DAYS	Weakness Lt Ul & Ll	hyperdense sss	hyperintense sss	sinus Thrombosis	good
23	30	NVD	PN 7 DAYS	Headache	hyperdense lt sigmoid sinus	hyperintense lt sigmoid sinus	sinus Thrombosis	good
24	31	LSCS	PN 2 DAYS	Headache	hyperdense sss	not done	sinus Thrombosis	good
25	19	NVD	PN 5 DAYS	Seizures	hyperdense sss, straight sinus	hyperintense sss, straight sinus	sinus Thrombosis	good
26	22	LSCS	PN 1 DAY	Weakness Lt Ul & Ll	rt parietal hagic infarct	rt parietal hagic infarct with cortical vein thrombosis	cortical vein thrombosis	good
27	23	NVD	PN 5 DYS	Headache	hyperdense lt sigmoid & transverse sinus	not done	sinus Thrombosis	good
28	21	LSCS	PN 2 DAYS	Headache , Weakness	lt frontal hagic infarct	lt frontal hagic infarct with cortical vein thrombosis	cortical vein thrombosis	good
29	20	LSCS	PN 1 DAY	Altered Sensorium	B/L frontoparietal white matter edema-PRES	PRES	PRES	good
30	23	LSCS	PN 2 DAYS	Weakness,Headache	rt temporal hagic infarct	rt temporal hagic infarct with cortical vein thrombosis	cortical vein thrombosis	good
31	19	AN	AN 7 MONTHS	Vomiting,Headache	not done	b/l parietooccipital white matter hyperintensity on T2/FLAIR	PRES	good
32	18	NVD	IMMEDIATE PN	Headache	hyperdense rt sigmoid & transverse sinus	hyperintense rt sigmoid & transverse sinus	sinus Thrombosis	good
33	22	LSCS	PN 2 DAYS	Weakness	lt parietal hagic infarct	lt parietal hagic infarct with cortical vein thrombosis	cortical vein thrombosis	good
34	24	LSCS	PN 1 DAY	Seizures	hyperdense sss	hyperintense sss	sinus Thrombosis	good
35	23	LSCS	PN 2 DAYS	altered sensorium	rt parietal hagic infarct	rt parietal hagic infarct with cortical vein thrombosis	cortical vein thrombosis	good
36	19	LSCS	PN 2 DAYS	non specific symptoms	hyperdense rt sigmoid & transverse sinus	hyperintense rt sigmoid & transverse sinus	sinus Thrombosis	good
37	20	LSCS	PN 1 DAY	vomiting	Lt frontoparietal white matter edema-PRES	b/l frontoparietal white matter hyperintensity on T2/FLAIR	PRES	good
38	22	NVD	PN 2 DAYS	Seizures,Weakness	lt parietal hagic infarct	lt parietal hagic infarct with cvt	cortical vein thrombosis	good
39	19	AN	AN 8 MONTHS	Headache	not done	hyperintense lt sigmoid sinus	sinus Thrombosis	good
40	21	LSCS	PN 3 DAYS	Seizures	hyperdense rt sigmoid,transverse & sss	hyperintense rt sigmoid,transverse & sss	sinus Thrombosis	good

S.No	Age	NVD/LSCS	AN/PN	Symptoms	CT findings	Mri findings	Diagnosis	Prognosis
41	22	NVD	PN 1 DAY	Blurred Vision,Headache	Lt frontoparietooccipital white matter edema	b/l frontoparietooccipital white matter hyperintensity on T2/FLAIR	PRES	good
42	21	NVD	PN 1 DAY	Altered Sensorium	ich right frontal lobe	ich right frontal lobe	PCA	good
43	22	LSCS	PN 3 DAYS	Seizures	hyperdense lt sigmoid,transverse & sss	hyperintense lt sigmoid,transverse & sss	sinus Thrombosis	good
44	19	NVD	PN 1 DAY	Status Epilepticus	hypendense sss,sigmoid & straight sinus	b/l thalamic hgic infarct with sinus thrombosis	sinus Thrombosis	bad
45	20	LSCS	PN 1 DAY	Headache	hyperdense lt sigmoid sinus	hyperintense lt sigmoid sinus	sinus Thrombosis	good
46	21	LSCS	PN 2 DAYS	Weakness,Seizures	hyperdense sss with rt parietal hagic infarct	hyperintense sss with rt parietal hagic infarct	sinus Thrombosis	good
47	19	LSCS	PN 1 DAY	Weakness Rt Ul & Ll	Lt parietal lobe hypodense area	not done	Arterial infarct	good
48	21	NVD	PN 2 DAYS	Vomiting,Headache	Lt parietooccipital white matter edema-PRES	b/l parietooccipital white matter hyperintensity on T2/FLAIR	PRES	good
49	23	AN	AN 8 MONTHS	Seizures	not done	hyperintense rt sigmoid & transverse sinus	sinus Thrombosis	good
50	19	NVD	PN 1 DAY	Weakness,Headache	rt frontal hagic infarct	rt frontal hagic infarct with CVT	cortical vein thrombosis	good

ABBREVIATIONS TO MASTER CHART

AN	-	Antenatal
PN	-	Postnatal
LSCS	-	Lower segment caesarean segment
NVD	-	Normal vaginal delivery
SSS	-	Superior sagittal sinus
CVT	-	Cerebral vein thrombosis
PRES	-	Posterior reversible encephalopathy syndrome
PCA	-	Postpartum cerebral angiopathy
HAGIC	-	Hemorrhagic
LT	-	Left side
RT	-	Right side
UL	-	Upper limb
LL	-	Lower limb
B/L	-	Bilateral
FLAIR	-	Fluid attenuation inversion recovery
T1W	-	T1 weighted